ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN CHRONIC ASYMPTOMATIC ALCOHOLICS

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CERTIFICATE

This is to certify that this dissertation entitled **"ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN CHRONIC ASYMPTOMATIC ALCOHOLICS"** submitted by Dr.PRIYA KUBENDIRAN to The Tamil Nadu Dr.M.G.R. Medical University Chennai is in partial fulfillment of the requirement for the award of M.D DEGREE BRANCH I (General medicine) and is a bonafide research work carried out by her under direct supervision and guidance.

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DECLARATION

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This dissertation is submitted to the Tamilnadu Dr.M.G.R Medical university towards the partial fulfillment of requirement for the award of M.D. DEGREE Branch-1in General Medicine.

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ABBREVIATIONS

LV – left ventricle

PR – pulse rate

BP – blood pressure

BSA – body surface area

FBS/PPBS – fasting / postprandial blood sugar

Hb – hemoglobin

AST – aspartate aminotransferase

ALT – alanine aminotransferase

GGT – gamma glutamyl transferase

TGL – triglycerides

LDL - low density lipoprotein

TSH – thyroid stimulating hormone

$LVID_d$ – left ventricular internal dimension in diastole

EDVI – end diastolic volume index

ESVI – end systolic volume index

FS – fractional shortening

EF – ejection fraction

PWT_d – posterior wall thickness in diastole

$IVST_d$ – interventricular septal thickness in diastole

LVMI – left ventricular mass index

IVRT – isovolumic relaxation time

DT – deceleration time

E – peak early transmitral flow velocity

A – peak later transmitral flow velocity

Introduction

INTRODUCTION

Alcohol abuse is prevalent across all countries. The pattern of alcohol use varies depending on age, religion, education and other socio-demographic characteristics. Since 1970, consumption of alcohol in developing countries and developed countries have increased by 47% and 35%, respectively[6] . Alcohol causes 4% of the total Disability Adjusted Life Years (DALYs) and alcohol abuse disorders account for 1.4% of the total burden of disease[7].

The prevalence of current alcohol use has been reported to be 20–38% in males and 10% among females[8]. Studies in northern India found the 1 year prevalence of alcohol use to be between 25 and 40%[9,10] . In southern India, the prevalence of current alcohol use varies between 33 and 50%, with a higher prevalence among the lesser educated and the poor[11] . These studies have shown that alcohol consumption rates are much higher among men than women.

Although one to two drinks per day in a healthy individual can have some beneficial cardiovascular effects, at higher doses alcohol is toxic to most organ systems. When ingested in higher amounts, ethanol may cause ventricular systolic and/or diastolic dysfunction, systemic arterial hypertension, arrhythmias and even

sudden cardiac death. Excess alcohol consumption over a long period can progressively worsen cardiac function and cause dilated cardiomyopathy[12] .As the disease progresses, subtle signs may be identified that precede the appearance of clinically evident cardiac dysfunction[13] . This is significant as early abstinence may reverse the left ventricular dysfunction[14]. Numerous studies have been done on the effect of chronic alcohol consumption on systolic and diastolic LV function, some of which showed impaired [13,15] whereas others showed well preserved LV systolic function[16-18]. Normal[18] and impaired LV filling[19,20] has also been reported.

Hence this study was conducted to assess the systolic and diastolic function of the left ventricle in asymptomatic alcoholics attending Stanley medical college and hospital.

Aim of the Study

AIM OF THE STUDY

1. To assess preclinical left ventricular dysfunction in chronic asymptomatic alcoholics using 2 dimensional & Doppler echocardiography.

2. To correlate the possible differences with the duration of alcohol intake.

Review Of Literature

REVIEW OF LITERATURE

Alcohol is consumed at some point of time by up to 80% of the population. Though at low doses it can have some beneficial effects, heavy repetitive drinking increases the risk of health problems in many organ systems and cuts short the life span by an estimated decade.

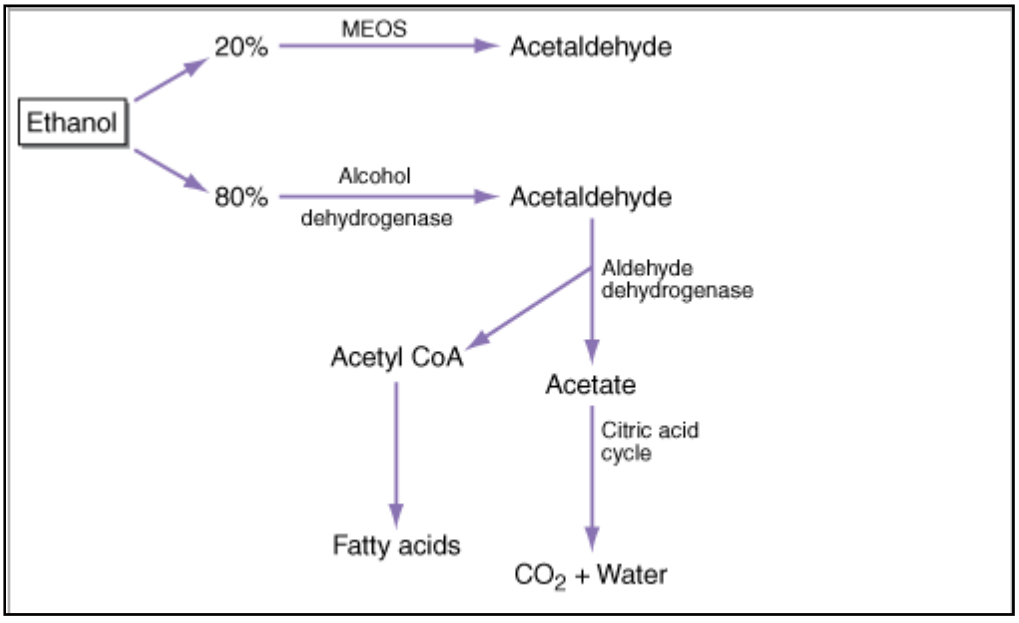
PHARMACOLOGY & METABOLISM

Ethanol, also called ethyl alcohol or pure alcohol, is a volatile, flammable, colourless liquid. It is a straight-chain alcohol, and its molecular formula is C_2H_5OH. It is a weakly charged molecule that moves easily through cell membranes, rapidly equilibrating between blood and tissues.

Ethanol is absorbed from mucous membranes of the mouth and esophagus in small amounts and from the stomach and large bowel in modest amounts. However, the major site of absorption is the proximal portion of the small intestine. The rate of absorption is increased by rapid gastric emptying; absence of congeners; absence of proteins, fats, or carbohydrates which interfere with

absorption; and by dilution to a modest percentage of ethanol (maximum at ~20% by volume).

Between 2% - 10% of ethanol is excreted directly through sweat, lungs or urine. But the major portion is metabolized to acetaldehyde, primarily in the liver. The most important pathway occurs in the cell cytosol where alcohol dehydrogenase (ADH) produces acetaldehyde, which is then rapidly converted by aldehyde dehydrogenase (ALDH) to acetate and acetyl CoA in the cytosol and mitochondria . A second pathway occurs in the microsomes of the smooth endoplasmic reticulum known as the microsomal ethanol-oxidizing system (MEOS) which is responsible for ≥ 10% of ethanol oxidation at high blood alcohol concentrations.



Alcohol supplies calories (70–100 kcal/drink) but they are devoid of minerals, vitamins and proteins. Alcohol interferes with absorption of nutrients like folic acid, pyridoxine (B_6), thiamine (B_1), nicotinic acid (niacin, B_3) and vitamin A and decreases their storage in the liver.

PATHOPHYSIOLOGY

At low or moderate doses, alcohol primarily acts as a positive allosteric modulator of $GABA_A$. Alcohol binds to several different subtypes of $GABA_A$. The main subtypes responsible for the subjective effects of alcohol are the α1β3γ2, α5β3γ2, α4β3δ and α6β3δ subtypes[21,22]. Activation of these receptors causes most of the effects of alcohol such as relaxation and relief from anxiety, sedation, ataxia and increase in appetite and lowering of inhibitions.

Alcohol has a powerful effect on glutamate as well. Alcohol decreases glutamate's ability to bind with NMDA receptors and acts as an antagonist of the NMDA receptor[23,24]. Hence, chronic alcohol users experience an upregulation of NMDA receptors because the brain is attempting to reestablish homeostasis. This alteration of receptor numbers is likely to be responsible for some of the symptoms seen in delirium tremens and alcohol withdrawal seizures.

Other targets such as sodium channels can also be affected by high doses of alcohol, and alteration in the numbers of these channels in chronic alcoholics is likely to be responsible for effects such as cardiac arrythmia[25].

Alcohol also affects opioid systems and cannabinol receptors, enhances activity of the dopamine-rich reward system and increases serotonin actions. It may also inhibit the uptake of adenosine.

TOLERANCE

3 types of tolerance can be seen :

(1) *Metabolic or pharmacokinetic tolerance:* This is seen after 1–2 weeks of daily drinking. There is an increase in the rate of hepatic ethanol metabolism upto 30%. This phenomenon disappears almost as rapidly as it develops.

(2) *Cellular or pharmacodynamic tolerance:* This develops through neurochemical changes that maintain relatively normal physiologic functioning despite the presence of alcohol. Withdrawal symptoms occur when there is a decrease in blood alcohol levels.

(3) *Behavioral tolerance* - Individuals learn to adapt their behavior so that they can function better than expected under influence of the drug.

BEHAVIORAL EFFECTS

The level of alcohol in the blood is expressed as milligrams or grams of ethanol per deciliter with blood values of about 0.02 g/dL resulting from the ingestion of one typical drink.

Effects of Blood Alcohol Levels in the Absence of Tolerance

Blood Level (g/dL)	Usual Effect
0.02	Decreased inhibitions, a slight feeling of intoxication
0.08	Decrease in complex cognitive functions and motor performance
0.20	Slurred speech, motor incoordination, irritability and poor judgement
0.30	Light coma and depressed vital signs
0.40	Death

ALCOHOL EQUIVALENTS

The amount of alcohol in grams of ethanol can be calculated as per the following conversion [26]:

30 ml hard liquor = 100 ml wine = 250 ml beer = 10 grams of ethanol which is usually considered as one drink. But the amount of pure ethanol in a standard drink varies from country to country.

THE EFFECTS OF ETHANOL ON ORGAN SYSTEMS

CARDIOVASCULAR SYSTEM

Although the ingestion of a moderate amount of ethanol (three to nine drinks/ week) appears to be associated with a reduced risk of cardiovascular disease, the consumption of excessive amounts has many adverse effects on the cardiovascular system.

SYSTEMIC ARTERIAL HYPERTENSION

Ethanol is of etiologic importance in up to 11 percent of men with hypertension. Individuals who consume more than 2 drinks daily are 1.5 to 2 times more likely to have hypertension when compared with age- and gender-matched nondrinkers[27]. This effect is dose related and is most prominent when the daily ethanol intake exceeds 30 gm[28] . Although the mechanism is unclear, previous studies have demonstrated that ethanol consumption increases plasma levels of

catecholamines, renin, and aldosterone which result in systemic arterial vasoconstriction. Abstinence results in normalization of the blood pressure.

LIPID METABOLISM

Ethanol inhibits the oxidation of free fatty acids by the liver, which stimulates hepatic triglyceride synthesis and the secretion of very low-density lipoprotein cholesterol. Hence, ethanol consumption results in hypertriglyceridemia. It also may increase the serum concentrations of total cholesterol and low-density lipoprotein (LDL). However, moderate alcohol consumption increases the serum concentration of high-density lipoprotein (HDL) cholesterol[29].

CORONARY ARTERY DISEASE

Consumption of large quantities of alcohol is associated with an increased incidence of atherosclerotic coronary artery disease and resultant cardiovascular morbidity and mortality. This may partly be due to the fact that heavy drinkers compared with non-drinkers have hypertension, an increased left ventricular muscle mass with concomitant diastolic and/or systolic dysfunction, and hypertriglyceridemia. In addition, they are often smokers.

However, mild to moderate ethanol intake (two to seven drinks/ week) appears to be associated with a decreased risk of cardiovascular morbidity and mortality in both men and women . Several prospectively performed cohort studies have demonstrated that drinkers of moderate amounts of ethanol are 40 to 70 percent less likely to manifest coronary artery disease when compared with nondrinkers or heavy consumers[30]. A meta-analysis of all experimental studies that assessed the effects of moderate alcohol intake on concentrations of high density lipoprotein cholesterol, apolipoprotein A I, fibrinogen, triglycerides and other biological markers previously found to be associated with risk of coronary heart disease, has concluded that 30 g of alcohol a day would cause an estimated reduction of 24.7% in the risk of coronary heart disease through changes in lipids and haemostatic factors[29]. Some studies have suggested that the consumption of all alcoholic beverages exerts such an effect, whereas others have reported that this cardioprotection is strongest with the consumption of wine[31].

The decrease in the cardiovascular risks is due to the beneficial effects of ethanol such as:

(a) an increase in the serum concentrations of HDL cholesterol and apolipoprotein A-I,

(b) inhibition of platelet aggregation,

(c) a decreased serum fibrinogen concentration,

(d) increased antioxidant activity (from the phenolic compounds and flavonoids contained in red wine), and

(e) improved fibrinolysis (due to increased concentrations of endogenous tissue plasminogen activator and a concomitant decrease in endogenous plasminogen activator inhibitor activity)

The reduction in the risk of coronary artery disease is similar among diabetic and non diabetic men and women[32]. In survivors of myocardial infarction, moderate ethanol consumption appears to reduce subsequent mortality[33].

ARRYTHMIAS

Ethanol consumption is associated with a variety of arrhythmias like atrial or ventricular premature beats, supraventricular tachycardia, atrial flutter or fibrillation, ventricular tachycardia or fibrillation. The most common arrhythmia is atrial fibrillation. Ethanol is of etiologic importance in about one third of subjects with new-onset atrial fibrillation. Most episodes occur after binge drinking, usually on weekends or holidays—known as the “holiday heart syndrome.”

Ethanol may be arrhythmogenic via several mechanisms. Concomitant factors like cigarette smoking, electrolyte disturbances, metabolic abnormalities, hypertension or sleep apnea may predispose to arrhythmias. Acute ethanol ingestion induces a diuresis, which is accompanied by urinary loss of sodium, potassium, and magnesium resulting in an electrolyte imbalance. The presence of myocardial interstitial fibrosis, ventricular hypertrophy, cardiomyopathy, and autonomic dysfunction also may enhance the likelihood of dysrhythmias. The treatment of these arrhythmias is abstinence from alcohol.

ALCOHOLIC CARDIOMYOPATHY

Alcoholic cardiomyopathy is a clinical diagnosis made in a patient presenting with a constellation of findings that includes a history of excessive alcohol intake, possible physical signs of alcohol abuse, heart failure, and supportive evidence consistent with dilated cardiomyopathy.

EFFECTS OF ETHANOL ON CARDIAC MYOCYTE STRUCTURE AND FUNCTION

Ethanol may cause myocardial damage via several mechanisms[34]. Ethanol and its metabolites, acetaldehyde and acetate, may exert a direct toxic effect on the myocardium or deficiencies of certain vitamins, minerals or electrolytes may

adversely affect myocardial function. Sometimes, certain toxic substances like lead or cobalt are added to alcoholic beverages which may be toxic to the myocardium.

Mechanisms of ethanol induced myocardial injury[2]:

Direct Toxic Effects
Uncoupling of the excitation/contraction system
Reduced calcium sequestration in sarcoplasmic reticulum
Inhibition of sarcolemmal ATP-dependent Na^+/K^+ pump
Reduction in mitochondrial respiratory ratio
Altered substrate utilization
Increased interstitial/extracellular protein synthesis
Toxic Effect of Metabolites
Acetaldehyde
Ethyl esters
Nutritional or Trace Metal Deficiencies
Thiamine
Selenium
Electrolyte Disturbances
Hypomagnesemia
Hypokalemia
Hypophosphatemia
Toxic Additives
Cobalt
Lead

Electron microscopic studies of the hearts of experimental animals with heavy ethanol ingestion demonstrate dilated sarcoplasmic reticula and swollen mitochondria with fragmented cristae and glycogen-filled vacuoles. With sustained exposure to ethanol, myofibrillar degeneration and replacement fibrosis appear. Microscopically, the hearts of chronic heavy consumers of ethanol manifest an increased accumulation of collagen in the extracellular matrix, as well as increased intermolecular cross-links.

EFFECTS ON CARDIAC FUNCTION

Chronic heavy ethanol ingestion may induce left ventricular diastolic and/or systolic dysfunction. Diastolic dysfunction which is partly due to interstitial fibrosis of the myocardium, is often demonstrable in chronic alcoholics even in the absence of symptoms or signs. About half of asymptomatic chronic alcoholics have echocardiographic evidence of left ventricular hypertrophy with preserved systolic performance. By Doppler echocardiography, the left ventricular relaxation time is often prolonged, the peak early diastolic velocity is reduced, and the acceleration of early diastolic flow is slowed which indicate left ventricular diastolic dysfunction[20,35].

Around 30 percent of asymptomatic chronic alcoholics have evidence of left ventricular systolic dysfunction in echocardiography. With continued heavy

ethanol ingestion, these subjects often develop dilated cardiomyopathy that manifests clinically as congestive cardiac failure. Ethanol abuse is the most common cause of non ischemic dilated cardiomyopathy. Women are more susceptible compared to males as they develop cardiomyopathy following ingestion of lesser quantities of ethanol[36].

Left ventricular systolic and diastolic function often improve following abstinence from alcohol[37]; these changes are more pronounced when abstinence is initiated early in the course of ethanol consumption.

There are individual variations in the susceptibility to ethanol's cardiotoxic effects. In this regard, some studies have suggested that genetic polymorphisms in the angiotensin-converting enzyme (ACE) gene may play a role in the development of ethanol-induced dilated cardiomyopathy. Subjects who are homozygous for the deletion polymorphism of the ACE gene (so-called *DD*) have increased plasma and cardiac levels of ACE. In the absence of ethanol consumption, these homozygotes are at increased risk of developing left ventricular hypertrophy and idiopathic dilated cardiomyopathy. Similarly, alcoholics who are homozygous for this deletion polymorphism are more likely to develop a dilated cardiomyopathy than alcoholic subjects without it[38]. Likewise, a polymorphism of the gene encoding the alcohol metabolizing enzyme, alcohol

dehydrogenase (*ALDH2*2*) increases the predilection for the development of alcoholic cardiomyopathy

QUANTITY OF ALCOHOL INTAKE IN CARDIAC DISEASE

The likelihood of developing an ethanol-induced dilated cardiomyopathy correlates with the amount of ethanol that is consumed in a lifetime. Most men who develop an ethanol-induced dilated cardiomyopathy have consumed more than 80 gm of ethanol per day for at least 5 years.A study by Urbano-Marquez et al[15] reported on 48 men with alcohol abuse with a mean daily intake of 243 g of alcohol and showed (1) an inverse relationship between total lifetime intake and ejection fraction and fractional shortening and (2) a direct relationship between total lifetime intake and LV mass. In persons who consumed 70 g of ethanol per day for 20 years, 36% had an abnormal ejection fraction.

DURATION OF ALCOHOL INTAKE IN CARDIAC DISEASE

Similarly, the likelihood of developing cardiomyopathy correlates with the duration of alcohol intake. A study by Lazarevic et al[39] demonstrated that the progression of left ventricular diastolic dysfunction was related to the duration of alcoholism.

EPIDEMIOLOGY

Sex related demographics

Although alcoholic cardiomyopathy is a disease that affects males more often than females due to a higher rate of alcohol abuse in men, females may be more susceptible to alcohol's cardiotoxic effects.

In 1995, Urbano-Marquez et al[36] described similar results in a study of 50 women and 100 men who abused alcohol. The authors reported a lifetime dose of alcohol in the female group that was 60% of that in the male group, but they found an equal incidence of cardiomyopathy in the males and females.

Age related demographics

Middle aged persons are most commonly affected and incidence is less in those younger than 40 years, although preclinical cardiac abnormalities have been demonstrated in young persons[20] engaging in chronic alcohol abuse.

CLINICAL FEATURES

Alcoholic cardiomyopathy similar to any dilated cardiomyopathy manifests as congestive cardiac failure. Dyspnoea, orthopnoea, and paroxysmal nocturnal dyspnoea are the hallmark complaints. Other symptoms like chest discomfort,

fatigue, palpitations, dizziness, syncope, anorexia may also be present. The onset of symptoms is usually insidious, but atrial fibrillation or any other tachyarrhythmia may precipitate acute decompensations.

On examination, the apical impulse may be displaced laterally implying cardiomegaly. Auscultation may reveal S_3, S_4, murmurs of mitral or tricuspid regurgitation. Pulmonary rales indicate pulmonary congestion secondary to elevated left heart pressures. Jugular venous distention, peripheral edema, and hepatomegaly are evidence of elevated right heart pressures.

However, before an individual manifests features of congestive cardiac failure, certain changes occur in the heart in the preclinical stage which is asymptomatic. There may be evidence of diastolic and/or systolic dysfunction which can only be picked up by echocardiography.

INVESTIGATIONS

Lab - Alcoholics may show an increase in mean corpuscular volume, aspartate aminotransferase and gamma glutamyl transferase.

Electrocardiogram– the following abnormalities may be seen : atrial fibrillation, flutter and premature contractions; conduction disturbances, such as degrees of atrioventricular block, right or left bundle-branch block, and hemiblocks;

prolonged QT interval; LV hypertrophy;nonspecific ST- and T-wave changes and Q waves.

Imaging - Chest radiographs usually show evidence of cardiac enlargement, pulmonary congestion, and pleural effusions.

Echocardiography :

Echocardiographic findings in persons with alcoholic cardiomyopathy, which are similar to those in persons with idiopathic dilated cardiomyopathy, are as follows:

- 4-chamber dilatation
- Globally decreased ventricular function
- Mitral and tricuspid regurgitation
- Pulmonary hypertension
- Evidence of diastolic dysfunction by Doppler
- Intracardiac thrombi (atrial or ventricular)
- LV hypertrophy

In the asymptomatic stage, echocardiography may show evidence of systolic and/ diastolic dysfunction. 2-D, M-mode and Doppler echocardiography are used. The following changes have been documented by numerous studies[15,19,39]:

- Increased chamber volumes (end systolic & end diastolic)
- Normal/reduced ejection fraction
- Increased left ventricular mass
- Increased LV posterior wall & interventricular septal thickness
- Diastolic dysfunction : prolonged isovolumic relaxation time
  Increased deceleration time
  Reduced E/A ratio

PROGNOSIS

The natural history of patients with alcoholic cardiomyopathy depends on each patient's ability to abstain from alcohol completely. Multiple case reports and small retrospective and prospective studies have clearly documented marked improvement or normalization of cardiac function with abstinence[37].

MANAGEMENT

The mainstay of therapy for alcoholic cardiomyopathy is complete abstinence from alcohol.

Medical therapy for symptomatic patients is identical to conventional therapy for other forms of heart failure. This includes treatment with an ACE inhibitors, digoxin, diuretics, beta blockers. Electrolyte abnormalities should be corrected promptly because of the risk of arrhythmia and sudden death. Anticoagulation should be initiated only after weighing the risks versus benefits. Thiamine (200 mg once daily), vitamin supplements, folic acid, and mineral supplementation are beneficial because of the significant prevalence of concomitant nutritional deficiencies in these patients.

NERVOUS SYSTEM

Around 35% of alcoholics experience a *blackout*, an episode of temporary anterograde amnesia, in which the person cannot recall what occurred during a drinking evening. Heavy drinking can also be associated with *a hangover syndrome* the following day characterized by thirst, nausea, vomiting, headache and fatigue.

Sleep disturbances are common. Although alcohol might initially help a person to fall asleep, it disrupts sleep throughout the rest of the night. The stages of sleep are also altered, and time spent in rapid eye movement (REM) and deep sleep

is reduced. Disturbing dreams are frequently experienced. Alcohol can cause snoring and exacerbate sleep apnea as it relaxes muscles in the pharynx.

Chronic high doses cause *peripheral neuropathy* in 5–15% of alcoholics. Symptoms are bilateral limb numbness, tingling, and paresthesias which are more prominent distally. Approximately 1% of alcoholics develop cerebellar degeneration or atrophy characterized clinically by nystagmus, unsteadiness and ataxia. Atrophy of the cerebellar vermis can be confirmed by neuroimaging. Rarely, in predisposed individuals (e.g. transketolase deficiency) thiamine deficiency may result in *Wernicke's* (ophthalmoparesis, ataxia, and encephalopathy) and *Korsakoff's* (retrograde and anterograde amnesia) *syndromes.*

Alcoholics can manifest *cognitive problems* lasting for weeks to months after an alcoholic binge. Many psychiatric syndromes can be seen temporarily during heavy drinking and subsequent withdrawal. These include an profound sadness lasting for days to weeks in the midst of heavy drinking (*alcohol-induced mood disorder*); temporary severe anxiety that often begins during alcohol withdrawal, and which can persist for a month or more after cessation of drinking (*alcohol-induced anxiety disorder*); and auditory hallucinations and/or paranoid delusions in a person who is alert and oriented (*alcohol-induced psychotic disorder*).

Studies have demonstrated that moderate alcohol consumption upto two drinks per day was significantly protective for ischemic stroke[40].

Once the brain has been repeatedly exposed to high doses of alcohol, any sudden decrease in intake can produce withdrawal symptoms like hand tremors, anxiety, agitation, tachycardia, tachypnea, fever, insomnia. These withdrawal symptoms generally begin within 5–10 h of decreasing ethanol intake, peak on the 2nd or 3rd day and improve by 4th or 5th day. A small percentage of alcoholics experience withdrawal seizures usually manifesting as a single generalized seizure.Very rarely, in alcohol dependent individuals, acute withdrawal may result in delirium (agitation, confusion and fluctuating levels of consciousness) associated with a tremor and autonomic overactivity known as *delirium tremens*.

THE GASTROINTESTINAL SYSTEM

Alcohol intake can result in esophagitis and gastritis which may manifest as gastrointestinal bleeding. A longitudinal tear in the mucosa at the gastroesophageal junction can be produced by violent retching or vomiting which is known as Mallory-Weiss lesion.

Acute pancreatitis is three times more common in alcoholics. There is a higher incidence of fatty liver because of impaired gluconeogenesis in the liver, resulting in a decrease in the amount of glucose produced from glycogen, increased lactate production, and decreased oxidation of fatty acids. These changes are reversible with abstinence but continued alcohol intake may lead to alcoholic hepatitis and later on, cirrhosis.

HAEMATOPOEITIC SYSTEM

Ethanol causes an increase in mean corpuscular volume of red blood cells due to its effects on stem cells. Concomitant folic acid deficiency manifests as hypersegmented neutrophils, decreased reticulocyte counts and a hyperplastic bone marrow. Chronic heavy drinking can decrease production of white blood cells, decrease granulocyte mobility and adherence, and impair delayed-hypersensitivity responses to novel antigens.

MALIGNANCIES

Alcohol increases the risk of the following cancers: mouth, pharynx and larynx, oesophagus, colorectum (men), breast (pre- and postmenopausal women). This is postulated to be due to the toxic effects of acetaldehyde, the breakdown product of ethanol metabolism.

REPRODUCTIVE SYSTEM

In males, consumption of modest doses of ethanol may increase sexual drive but they reduce the erectile capacity. Even in the absence of liver impairment, a minor percentage of chronic alcoholic men show irreversible testicular atrophy with shrinkage of the seminiferous tubules, decrease in ejaculate volume and a lower sperm count.

Chronic alcohol consumption in females can result in amenorrhea, a decrease in ovarian size, absence of corpora lutea with associated infertility and an increased risk of spontaneous abortion. Ethanol and acetaldehyde are transferred across the placental barrier which may result in *fetal alcohol syndrome* characterized by: facial abnormalities with epicanthal eye folds; poorly formed ear concha; small teeth with faulty enamel; atrial or ventricular septal defects; an aberrant palmar crease and limitation in joint movement; and microcephaly with mental retardation.

MUSCULOSKELETAL SYSTEM

Around 50 to 65 % manifest acute *alcoholic myopathy*, which is characterized by skeletal muscle weakness which may improve with abstinence, but some residual weakness usually remains. Chronic consumption of alcohol affects the

skeletal system by altering calcium metabolism, reducing growth in the epiphyses and decreasing the bone density that results in increased risk for fractures.

ENDOCRINE SYSTEM

Hormonal changes include a reduction in the levels of serum thyroxine (T_4) and triiodothyronine (T_3) and an increase in cortisol levels. There is inhibition of vasopressin secretion at high blood alcohol concentrations and enhanced secretion at low blood alcohol concentrations. Hormone irregularities should be re evaluated after a month of abstinence.

ALCOHOLISM

Alcoholism is a common problem worldwide. A recent National Household Survey of Drug Use in India[41] recorded alcohol use in the past year in 21% of adult males. The prevalence of current use of alcohol ranged from a low of 7% in the western states to 75% in the North-eastern states. Prevalence among women has been estimated at less than 5% but is much higher in the North-eastern states. Significantly higher use has been recorded among tribal, rural and lower socio-economic urban sections.

When repeated problems in multiple spheres of life develop due to alcohol consumption, the individual is likely to meet criteria for alcohol abuse or dependence. Worldwide, the diagnostic criteria for alcohol abuse and dependence of two institutions, the World Health Organization (WHO) and the American Psychiatric Association (APA) are the most widely accepted. The WHO issues the International Classification of Diseases (ICD) and the APA issues the Diagnostic and Statistical Manual of Mental Disorders (DSM). Both institutions regularly update their classification systems. ICD is in its 10th version (ICD-10) and DSM in its 4th (DSM-IV).

Definitions

DSM-IV[42]

ALCOHOL ABUSE

1. A maladaptive pattern of alcohol abuse leading to clinically significant impairment or distress, as manifested by *one or more* of the following, occurring *within a 12-month period*:
    - Recurrent alcohol use resulting in failure to fulfil major role obligations at work or home (e.g., repeated absences or poor work

performance related to substance use; substance-related absences; or neglect of children or household).

- Recurrent alcohol use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine).
- Recurrent alcohol-related legal problems (e.g., arrests for alcohol-related disorderly conduct).
- Continued alcohol use despite persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the alcohol (e.g., arguments with spouse about consequences of intoxication or physical fights).

2. These symptoms must never have met the criteria for alcohol dependence.

ALCOHOL DEPENDENCE

A maladaptive pattern of alcohol use, leading to clinically significant impairment or distress, as manifested by *three or more* of the following seven criteria, occurring at any time in the same *12-month period*:

1. Tolerance, as defined by either of the following:
    - A need for markedly increased amounts of alcohol to achieve intoxication or desired effect.

- Markedly diminished effect with continued use of the same amount of alcohol.

2. Withdrawal, as defined by either of the following:
    - The characteristic withdrawal syndrome for alcohol
    - Alcohol is taken to relieve or avoid withdrawal symptoms.
3. Alcohol is often taken in larger amounts or over a longer period than was intended.
4. There is a persistent desire or there are unsuccessful efforts to cut down or control alcohol use.
5. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol or recover from its effects.
6. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
7. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the alcohol (e.g., continued drinking despite recognition that an ulcer was made worse by alcohol consumption).

ICD – 10[45]

F 10 Mental and behavioural disorders due to the use of alcohol

The following fourth-character subdivisions are for use:

.0 Acute intoxication

.1 Harmful use

.2 Dependence syndrome

.3 Withdrawal state

.4 Withdrawal state with delirium

.5 Psychotic disorder

.6 Amnesic syndrome

.7 Residual and late onset psychotic disorder

.8 Other mental and behavioural disorders

.9 Unspecified mental and behavioural disorders

GENETICS OF ALCOHOLISM

Several separate and distinct characteristics appear to contribute to the risk of alcoholism. Some families are at risk for both alcoholism and drug dependence associated with the characteristic of high levels of impulsivity, as can be seen in the antisocial personality disorder. In other families, the risk for both alcohol and drug dependence may relate to a genetic vulnerability to schizophrenia, panic

disorder, or manic depressive disease. In certain other families, there is an increased risk for alcoholism alone due to a low response to alcohol and subsequently drinking higher doses to achieve the desired effects. In contrast, a decreased risk for heavy drinking can result from a mutation that causes the production of an inactive form of the enzyme aldehyde dehydrogenase. This results in a more intense response to alcohol due to higher levels of acetaldehyde following alcohol ingestion.

IDENTIFICATION OF THE ALCOHOLIC

The clinical diagnosis of alcohol abuse or dependence ultimately rests on the documentation of a pattern of repeated difficulties associated with alcohol use; the definition is not based on the quantity and frequency of alcohol consumption.

Physical signs and symptoms that can be useful in identifying alcoholism include mild and fluctuating hypertension, repeated infections such as pneumonia and otherwise unexplained cardiac arrhythmias. Other disorders suggestive of dependence include cirrhosis, unexplained hepatitis, pancreatitis, bilateral parotid gland swelling, peripheral neuropathy and malignancies of the head and neck, esophagus or stomach.

Laboratory tests that are likely to be abnormal in individuals consuming more than 6 drinks/day are gamma-glutamyl transferase (GGT) (>35 U) and carbohydrate-deficient transferrin (CDT) (>20 U/L). The sensitivity and specificity is ≥ 70%. The combination of both the tests is likely to be more accurate. These serologic markers can also be useful in monitoring abstinence, as they are likely to return toward normal within several weeks of the cessation of drinking. Other investigations that are abnormal are high-normal MCVs (≥91μ m^3) and serum uric acid (>7 mg/dL).

Disorders related to the use of alcohol are common in general hospitals and are associated with higher morbidity and mortality. However, between 37% and 49% of the patients suffering from alcohol-related disorders and admitted to general hospitals due to physical illnesses are not identified as such. This is partially due to time constraints, fear of discrimination and lack of awareness of the importance of the diagnosis on the part of the patient and the attending physician. Therefore,to facilitate the screening of such patients, diagnostic instruments that are brief, simple, easy to apply and unintimidating like CAGE and AUDIT have been developed.

CAGE

A clinically helpful approach to diagnosis of alcohol dependence and abuse is the use of the CAGE questionnaire:

Acronym	Question
C	Have you ever felt you ought to ***C***ut down on your drinking?
A	Have people ***A***nnoyed you by criticizing your drinking?
G	Have you ever felt ***G***uilty or bad about your drinking?
E	Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover (***E***yeopener)?

One "yes" response should raise suspicion of an alcohol use problem, and more than one is a strong indication that abuse or dependence exists.

A study by Castells MA et al[44] showed that the CAGE questionnaire presented its highest sensitivity (93.8%) when the cut-off point of 0/1 (one or more “positive” responses indicating a positive test) was used. The specificity for this cut-off point was 85.5%.

Another study by Malet et al[45] concluded that The CAGE questionnaire for a cut-off of 2 had a sensitivity of 77% and a specificity of 94%. The CAGE test was more sensitive for patients diagnosed as alcohol-dependent than for alcohol abusers (61% vs. 84%) with the same specificity (94%). The eye-opening question (E) differentiated sharply between abuse and dependency, with sensitivities of 18% and 46%, respectively.

AUDIT

The AUDIT (Alcohol Use Disorders Identification Test)[48] was developed by the WHO as a simple method of screening for excessive drinking and to assist in brief assessment.

The AUDIT helps the practitioner to identify whether the person has hazardous (or risky) drinking, harmful drinking or alcohol dependence.

Hazardous drinking is a pattern of alcohol consumption that increases the risk of harmful consequences for the user or others.

Harmful use refers to alcohol consumption that results in consequences to physical and mental health. Some also consider social consequences among the harms caused by alcohol.

Alcohol dependence is a cluster of behavioural, cognitive, and physiological phenomena that may develop after repeated alcohol use.

The AUDIT was developed and evaluated over a period of two decades and it has been found to provide an accurate measure of risk across gender, age and cultures. It is consistent with ICD-10 definitions of alcohol dependence and harmful alcohol use.

AUDIT Questionnaire

1.How often do you have a drink containing alcohol? *Never (0) to 4+ per week (4)*

2. How many drinks containing alcohol do you have on a typical day?

1 or 2 (0) to 10+ (4)

3.How often do you have six or more drinks on one occasion?

Never (0) to daily or almost daily (4)

4. How often during the last year have you found that you were not able to stop drinking once you had started? *Never (0) to daily or almost daily (4)*

5. How often during the last year have you failed to do what was normally expected from you because of drinking? *Never (0) to daily or almost daily (4)*

6. How often during the last year have you needed a first drink in the morning to get yourself going after a heavy drinking session?

Never (0) to daily or almost daily (4)

7. How often during the last year have you had a feeling of guilt or remorse after drinking? *Never (0) to daily or almost daily (4)*

8. How often during the last year have you been unable to remember what happened the night before because you had been drinking?

Never (0) to daily or almost daily (4)

9. Have you or someone else been injured as a result of your drinking?

No (0) to yes, during the last year (4)

10. Has a relative, friend, doctor or other health worker been concerned about your drinking or suggested that you should cut down?

No (0) to yes, during the last year (4)

Scores for each question range from 0 to 4 with the first response for each question being zero, the second scoring 1 and so on. For questions 9 and 10 the scoring is 0,2,4.

Interpretation : ≥ 8 - harmful / hazardous drinking

≥ 13 in women
≥ 15 in men
} Alcohol dependence

ASSESSMENT OF CARDIAC FUNCTION[3-5]

Echocardiography is an excellent tool for noninvasive assessment of the left ventricle.

SYSTOLIC FUNCTION

Echocardiography can measure several parameters as an expression of systolic function of the heart.

Measurements of the left ventricular cavity dimension and wall thickness can be readily derived from M-mode recordings (figure 1) in parasternal long axis view at the level of mitral valves and are usually made according to the recommendations of the American Society of Echocardiography (ASE) at end diastole (the onset of the QRS complex) and end systole (the point of maximum upward motion of the LV posterior wall endocardium)[47].

Cardiac chamber volumes (end systolic and end diastolic) and ejection fraction are measured by 2-dimensional echocardiography (figure 2). Left ventricular volumes is calculated based on Simpson's rule, which derives measurements by dividing the LV by parallel planes into a number of small segments and then summating the area of the individual disks.

LV MASS

Is calculated using the formula:

LV mass (grams) = $\mathbf{0.8 \{ 1.04 [(LVID + IVST + PWT)^3 - LVID^3] \} + 0.6}$

Where $LVID_d$ – left ventricular internal diameter in diastole (cm)

$IVST_d$ – interventricular septal thickness in diastole (cm)

PWT_d – posterior wall thickness in diastole (cm)

DIASTOLIC FUNCTION

Doppler echocardiography is used for evaluation of ventricular diastolic filling. The transmitral velocity curves reflect the relative pressure gradients between the left atrium and ventricle throughout diastole. They are influenced by the rate of ventricular relaxation, the driving force across the valve, and the compliance of the ventricle. Doppler recordings of transmitral flow velocities are recorded at the level of leaflet tips in apical four chamber view.

Figure 3 illustrates the left ventricular inflow velocities and measurements for assessing the diastolic function of the left ventricle. Figure 3(A) shows the normal situation. The early inflow of blood reaches a peak at the E point. Flow then

decelerates until atrial systole, at which time the left atrial pressure rises above the left ventricular pressure and flow again passes through the mitral valve.

Alterations in left ventricular diastolic function may reduce the height of the E wave and increase the height of the ‘A’ wave. This type of abnormality is usually accompanied by prolongation of the isovolumic relaxation time (IVRT) and prolongation of the decelaration time (DT). [Figure 3 (B)] The hemodynamic abnormalities responsible for this pattern usually are reduced left ventricular relaxation and slower fall in left ventricular pressure.

The other pathologic pattern that is seen with mitral flow velocities is the reverse – a tall E wave and a short A wave. This pattern is accompanied by short isovolumic relaxation and deceleration times. [Figure 3 (D)] This type of mitral inflow can be produced by elevated left ventricular filling pressures. With elevated early diastolic pressure, the flow into the left ventricle is accelerated and there may be relatively little blood to propel with atrial systole.

In some disease states, the initial pathologic pattern in abnormal relaxation with a short E wave and tall A wave. If mitral regurgitation or congestive heart failure raises the left ventricular filling pressure, then the pattern may reverse and

the E wave will become taller and A wave will become shorter. Thus a transition situation can occur whereby ‘pseudonormalisation’ of the mitral inflow can occur. [Figure 3 (C)].

Materials and Methods

MATERIALS AND METHODS

1. Type of Study : Observational Study

2. Place : Stanley medical college and hospital, Chennai.

3. Collaborating Departments : Department of Cardiology.

4. Duration of Study : April 2011 to September 2011

5. Ethical clearance : Ethical clearance was obtained and the study was initiated

6. Consent : Informed consent was obtained before taking up the case for study

7. Inclusion Criteria:

50 alcoholics attending medicine department who satisfied the following criteria were enrolled in the study

- Positive diagnostic criteria for alcohol abuse or dependence as established by DSM IV criteria
- Age $\leq$ 60 years
- Daily ethanol consumption $\geq$ 90 grams $\geq$ 4 days/week
- Drinking history $\geq$ 5 years
- No history of any systemic diseases

8.Exclusion criteria :

- Ischemic/ rheumatic/ congenital heart disease
- Diabetes mellitus
- Hypertension
- Hyperlipidemia
- Thyroid disorders

9. Methods

The alcoholics who satisfied the criteria for alcohol abuse or dependence according to DSM IV criteria[42] were selected.

A detailed history regarding the duration and amount of alcohol consumption was obtained. The amount of alcohol consumed was then converted to grams of ethanol as per the following conversions[26] :

Alcohol equivalents:

30 ml spirits = 100 ml wine = 250 ml beer = 10 grams of ethanol

The alcoholics were then divided into three groups as follows, based on the duration of alcohol consumption, in order to evaluate possible differences in the left ventricular function in relation to the duration of drinking.

Group 1 (Short) : 5 – 9 years

Group 2 (Intermediate) : 10 – 15 years

Group 3 (Long) : > 15 years

A history of diabetes, hypertension, ischemic heart disease, rheumatic heart disease, hyperlipidemia, thyroid disorders and any other systemic disease were ruled out.

General physical examination, vital signs and systemic examination was done. Height and weight were obtained in order to calculate body surface area using **MOSTELLER'S FORMULA :**

$$BSA\ (m^2) = ([Height(cm) \times Weight(kg)]/ 3600)^{½}$$

The following investigations were done :

- Complete blood count
- Renal function tests
- Fasting and postprandial blood sugar

- Fasting lipid profile
- Thyroid function tests
- Liver function tests
- Electrocardiogram
- Chest x ray
- Echocardiography

ECHOCARDIOGRAPHY

Echocardiography was done using Mylab Easovite and Aloka systems.

The parameters studied using 2 dimensional, M –mode and Doppler echocardiograph were :

- Left ventricular internal dimension in diastole ($LVID_d$)
- End diastolic volume (EDV)
- End systolic volume (ESV)
- Interventricular septal thickness in diastole ($IVST_d$)
- Left ventricular posterior wall thickness in diastole (PWT_d)
- Ejection Fraction (EF)
- Left ventricular mass
- Isovolumic relaxation time (IVRT)

- Deceleration time (DT)
- Transmitral E and A velocities
- E/A ratio

The end systolic and diastolic volumes; and LV mass were converted to indices by dividing by body surface area.

10. Statistical analysis

Computer analysis of the data was done using SPSS Version 19 software. The range, mean and standard deviations were calculated. Analysis of variance was used to compare the differences among the means of alcoholics and controls. The three alcoholic groups were compared individually with the controls by unpaired t – test. Stepwise multiple linear regression analysis was used to evaluate the effects of several independent variables on the echocardiographic parameters in alcoholics. Significance was established at $p < 0.05$.

Observations and Results

OBSERVATIONS AND RESULTS

The results of the present study are presented in the ensuing pages.

Table 1 : PATIENT'S CHARACTERISTICS

	Controls	**Group 1**	**Group 2**	**Group 3**
	n = 50	n = 18 (36%)	n = 19 (38%)	n = 13 (26%)
Age (years)	39.26 ± 8.98	33.33 ± 6.98	38.26 ± 3.62	50.15 ± 4.99
Smokers	24 (48%)	11 (61%)	8 (42%)	7 (53%)
Duration of drinking (yrs)	-	7.80 ± 1.07	11.47 ± 1.22	19.04 ± 2.31
Ethanol consumption (g/day)	-	114.44 ± 27.70	123.16 ± 18.27	190 ± 38.08
Height (cm)	165.42 ± 6.22	166.06 ± 6.45	164 ± 7.09	167.30 ± 7.75
Weight (kg)	67.82 ± 9.46	67.39 ± 11.84	78.11 ± 7.48	77.62 ± 15.33
BSA (m^2)	1.76 ±0.15	1.76 ± 0.18	1.79 ± 0.17	1.89 ± 0.22
Pulse rate (beats/min)	79.16 ± 7.14	81.67 ± 6.95	79.53 ± 8.49	81.15 ± 5.55
Systolic BP (mm Hg)	122.24 ± 6.83	124.56 ± 5.81	124 ± 7.09	123.54 ± 5.95
Diastolic BP (mm Hg)	77.52 ± 5.56	78 ± 3.88	78.11 ± 5.48	76.77 ± 5.20

The data are presented as mean ± SD or percentage of patients. The baseline characteristics of controls and alcoholics were comparable.

Table 2 : LABORATORY DATA

	Controls	**Group 1**	**Group 2**	**Group 3**
Hb (g/dL)	12.73 ± 0.51	12.63 ± 0.60	12.42 ± 0.53	12.89 ± 0.60
AST (U/L)	26.34 ± 6.15	52.78 ± 15.13	66.89 ± 15.94	89.15 ± 14.25
ALT (U/L)	27.02 ± 6.48	45.50 ± 12.45	58.32 ± 15.52	49.38 ± 28.04
GGT (U/L)	27.32 ± 7.83	63.55 ± 10.94	67 ± 12.80	84.15 ± 14.32
T.Bilirubin (mg/dL)	0.80 ± 0.21	0.84 ± 0.22	0.87 ± 0.15	0.82 ± 0.12
T.Cholesterol (mg/dL)	165.24 ± 13.43	165 ± 18.85	153.26 ± 18.01	163.77 ± 20.67
TGL (mg/dL)	126.90 ± 9.85	125.89 ± 9.63	118.74 ± 16.22	121.69 ± 10.96

The data are presented as mean ± SD.

The labaratory parameters were similar in controls and cases except that alcoholics showed elevations in liver enzymes (AST, ALT, GGT) which was significant :

AST	p = 0.000
ALT	p = 0.027
GGT	p = 0.000

The echocardiographic changes in alcoholics compared to controls are presented in the following pages :

2-D AND M-MODE ECHOCARDIOGRAPHIC MEASUREMENTS

Table 3 : EDVI & ESVI

The end diastolic and end systolic volume indices of controls and alcoholics are presented below :

		Range	Mean	Std. Deviation	
EDVI (mL/m^2)	**Controls**	34.55 – 60.38	47.04	5.73	**p = 0.000**
	Group 1	40.12 – 56.77	47.43	4.78	
	Group 2	46.03 – 61.53	51.50	4.53	
	Group 3	48.56 – 65.43	54.86	4.91	
ESVI (mL/m^2)	**Controls**	11.11 – 18.86	14.18	1.69	**p = 0.899**
	Group 1	11.11 – 17.03	14.29	1.59	
	Group 2	11.73 – 18.86	14.30	1.75	
	Group 3	11.17 – 16.27	13.87	1.83	

The mean end diastolic volume index was significantly higher in alcoholics (**$p<0.001$**) compared to controls.

Table 4 : EJECTION FRACTION & LEFT VENTRICULAR MASS INDEX

		Range	Mean	Std. Deviation	
EF (%)	**Controls**	64 - 86	74.04	5.75	**p = 0.688**
	Group 1	65 - 81	74.44	4.68	
	Group 2	67 - 87	75.68	5.66	
	Group 3	67 - 85	75.31	5.39	
LVMI (g/m^2)	**Controls**	53.10 – 101.92	75.73	10.66	**p = 0.000**
	Group 1	63.30 – 85.45	75.07	7.18	
	Group 2	66.57 – 95.43	81.93	7.55	
	Group 3	81.63 – 104.50	94.44	6.95	

There was no statistically significant difference in mean ejection fraction between the two groups.

The estimated mean left ventricular mass index was higher in alcoholics which was statistically significant (**p<0.001**).

Table 6 : POSTERIOR WALL THICKNESS & INTERVENTRICULAR SEPTAL THICKNESS

		Range	Mean	Std. Deviation	
PWT_d (cm)	**Controls**	0.7 – 1.2	0.89	0.10	**p = 0.000**
	Group 1	0.7 – 1.1	0.86	0.11	
	Group 2	0.8 - 1	0.92	0.08	
	Group 3	0.9 – 1.2	1.06	0.09	
$IVST_d$ (cm)	**Controls**	0.7 – 1.2	0.93	0.12	**p = 0.128**
	Group 1	0.8 – 1.2	0.98	0.11	
	Group 2	0.8 – 1.1	0.98	0.10	
	Group 3	0.7 – 1.2	1.00	0.13	

The posterior wall thickness in diastole (**p<0.001**) showed a significant increase in alcoholics.

DOPPLER LEFT VENTRICULAR DIASTOLIC FILLING VARIABLES

Table 7 : ISOVOLUMIC RELAXATION TIME & DECELERATION TIME

		Range	Mean	Std. Deviation	
IVRT (ms)	**Controls**	63 - 127	85.04	13.33	**p = 0.000**
	Group 1	70 - 93	76.72	5.21	
	Group 2	74 - 100	87.11	6.44	
	Group 3	75 - 120	96.98	12.74	
DT (ms)	**Controls**	113 - 220	160.36	24.25	**p = 0.000**
	Group 1	132 - 185	153.41	15.35	
	Group 2	142 - 196	171.63	15.40	
	Group 3	172 - 226	198.42	15.70	

The isovolumic relaxation time and the deceleration time of the early transmitral flow velocity was significantly prolonged in alcoholics compared to controls (**p<0.001**).

Table 8 : E, A & E/A

		Range	Mean	Std. Deviation	
E (m/s)	**Controls**	0.63 – 1.12	0.85	0.12	**p = 0.132**
	Group 1	0.53 – 1.02	0.83	0.16	
	Group 2	0.60 – 1.03	0.80	0.13	
	Group 3	0.50 – 1.03	0.76	0.16	
A (m/s)	**Controls**	0.40 – 0.90	0.67	0.13	**p = 0.000**
	Group 1	0.40 – 0.91	0.61	0.15	
	Group 2	0.52 – 0.92	0.70	0.12	
	Group 3	0.50 – 1.12	0.87	0.17	
E/A	**Controls**	0.79 – 1.92	1.30	0.29	**p = 0.000**
	Group 1	1.09 – 1.68	1.37	0.17	
	Group 2	0.92 – 1.38	1.15	0.14	
	Group 3	0.67 – 1.22	0.89	0.18	

The peak early transmitral flow velocity (E) did not show a statistically significant difference among alcoholics and controls. The peak late transmitral flow velocity (A) was higher in alcoholics (**p<0.001**). E/A ratio was significantly lower in alcoholics (**p<0.001**) compared to controls.

CORRELATION WITH DURATION OF ALCOHOLISM

Stepwise multiple linear regression analysis was used to evaluate the effects of several independent variables (age, pulse rate, blood pressure, duration of alcoholism, quantity of ethanol consumption) on the echocardiographic parameters, each of which was considered separately as a dependent variable.

The left ventricular end diastolic volume index (**p=0.000**), LV mass index (**p=0.000**), posterior wall thickness (**p=0.000**), isovolumic relaxation time (**p=0.002**), deceleration time (**p=0.020**) and A (**p=0.012**) were significantly related to the duration of alcohol consumption.

The ejection fraction and interventricular septal thickness were not affected by any of the variables mentioned above.

EARLIEST CHANGE

The earliest abnormality that occurred was detected by comparing the three alcoholic groups individually with controls by t-test.

Table 9 : Comparison between the means of individual alcohol groups and controls

PARAMETER	GROUP 1	GROUP 2	GROUP 3
EDVI	p = 0.793	**p = 0.003***	**p = 0.000***
ESVI	p = 0.824	p = 0.802	p = 0.563
EF	p = 0.790	p = 0.291	p = 0.476
LVMI	p = 0.807	**p = 0.023***	**p = 0.000***
PWT	p = 0.170	p = 0.349	**p = 0.000***
IVST	p = 0.178	p = 0.112	p = 0.061
IVRT	p = 0.632	**p = 0.012***	**p = 0.008***
DT	p = 0.261	**p = 0.031***	**p = 0.000***
E	p = 0.509	p = 0.220	p = 0.132
A	p = 0.097	p = 0.244	**p = 0.000***
E/A	p = 0.397	p = 0.021	**p = 0.000***

* $p < 0.05$

There was no significant difference between group 1and controls. Comparison between group 2 and controls showed an increase in end diastolic volume index,

left ventricular mass, deceleration time and isovolumic relaxation time. Comparison between group 3 and controls demonstrated a significant increase in posterior wall thickness & A; and a lower E/A ratio, in addition to the forementioned differences.

Discussion

DISCUSSION

Chronic alcohol consumption leads to dilated cardiomyopathy in the long run. However, before clinical symptoms appear, certain changes occur in the heart which can be detected by echocardiography. These changes may be reversible with early abstinence. Hence it is significant to detect such changes. Many studies have been done in this regard which have shown conflicting results. Thus, this study was undertaken with a view to detect the cardiac abnormalities in chronic asymptomatic alcoholics.

50 alcoholics were included in this study after applying the selection criteria. They were divided into 3 groups based on the duration of drinking in order to assess any difference in left ventricular function relative to the duration of drinking. 50 normal individuals with similar baseline characteristics were included as controls.

The number of alcoholics in each group were :

Group 1 - 18 (36%)

Group 2 - 19 (38%)

Group 3 - 13 (26%)

PATIENT CHARACTERISITICS

The baseline characteristics like age, body surface area, pulse and blood pressure of the cases and controls were comparable.

The duration of alcohol consumption varied from 6 to 23 years. The mean ethanol consumption was 137.4 ± 41.84 grams per day. The mean ethanol consumption was highest (190 ± 38.08 grams) in the group with the longest duration of drinking. This can be attributed to the development of tolerance.

The laboratory parameters showed no significant differences among controls and alcoholics except for liver function tests, where alcoholics showed a mild elevation in enzymes (gamma glutamyl transferase, aspartate & alanine aminotransferases) which could be due to chronic ethanol consumption.

ECHOCARDIOGRAPHIC MEASUREMENTS

The following table summarizes the results of our study which is compared with a similar study by Lazarevic et al[39].

Lazarevic et al assessed the left ventricular function in 89 chronic asymptomatic alcoholics. He had divided the alcoholics into 3 groups (short, intermediate and long) according to the duration of alcohol consumption.

	Our study				**Lazarevic et al**			
	Controls	**Group 1**	**Group 2**	**Group 3**	**Controls**	**Short**	**Intermed-iate**	**Long**
Duration (years)	-	7.80	11.47	19.04	-	8	13	19
Amount (grams)	-	114.44	123.16	190	-	258	252	247
EF	74.04	74.44	75.68	75.31	67	66	67	66
EDVI	47.04	47.43	51.50	54.86	65	74	72	72
ESVI	14.18	14.29	14.30	13.87	21	25	24	25
LVMI	75.73	75.07	81.93	94.44	79	86	94	93
PWT_d	0.89	0.86	0.92	1.06	0.83	0.88	0.94	0.91
$IVST_d$	0.93	0.98	0.98	1.00	0.89	0.86	0.90	0.91
IVRT	85.04	76.72	87.11	96.98	83	90	93	92
DT	160.36	153.41	171.63	198.42	170	167	185	188
E	0.85	0.83	0.80	0.76	0.59	0.55	0.59	0.54
A	0.67	0.61	0.70	0.87	0.43	0.42	0.47	0.53
E/A	1.30	1.37	1.15	0.89	1.40	1.36	1.31	1.08

The data are presented as mean values.

The left ventricular systolic function and chamber dimensions were assessed using 2-D and M-mode echocardiography.

Our study found no statistically significant difference in ejection fraction between controls and alcoholics which is similar to the observations made by Lazarevic et al[39] and Askanas et al[48].

A significant increase in the end diastolic volume index in alcoholics compared to controls was observed in this study. There was no significant difference in end systolic volume index. However, Lazarevic et al[39] reported increased left ventricular systolic and diastolic dimensions. The normal end systolic volume index in our study may be explained by Frank Starling's law. An increase in end diastolic volume leads to increased contractility of the heart resulting in an increased stroke volume and reduced end systolic volume. The increased LV chamber volumes have been validated in animal studies by Shann D Kim et al[51] (in rodents) and by Nancy Morris et al[52] (in chickens).

The left ventricular mass index and posterior wall thickness was increased in alcoholics in this study. Interventricular septal thickness did not show a significant

difference. This was similar to the observations made by AM Lazarevic et al[39] , Kupari et al[19] and Mathews et al[50].

The diastolic function was assessed using Doppler echocardiography.

Our study demonstrated a significant increase in isovolumic relaxation time, a prolonged deceleration time and a lower E/A ratio in alcoholics. An increase in peak late transmitral flow velocity (A) was also observed. These observations imply impaired left ventricular relaxation or mild diastolic dysfunction.

A similar observation was stated by Lazarevic et al[39]. It has also been reported in studies by Kupari et al[19] and De Castro et al[53]. The delayed relaxation may be due to a delay in inactivation of actin myosin interaction because of impaired transport of calcium ions from sarcoplasma into the sarcoplasmic reticulum (Noren et al[54]). In addition, associated ventricular dilatation or myocardial hypertrophy may be responsible for delayed relaxation[55].

CORRELATION WITH THE DURATION OF ALCOHOLISM

The data of this study was analysed to assess the correlation between the echocardiographic changes and independent variables like age, pulse rate, blood pressure, duration of drinking and amount of alcohol consumed. The echocardiographic parameters showed a significant correlation with the duration of drinking. The end diastolic volume index, left ventricular mass index, posterior wall thickness, isovolumic relaxation time, deceleration time and peak late transmitral flow velocity demonstrated a significant increase as the duration of alcohol consumption increased.

Only a few studies have been done in this regard. Lazarevic et al[39] reported that alcoholics with a longer duration of drinking showed a longer deceleration time, higher A and reduced E/A. Kupari et al[35] observed that the posterior wall thickness was weakly related to the duration of heavy drinking. However, the LV hypertrophy and dysfunction were poorly related to the duration and severity of alcohol consumption. These discrepancies may be explained by the differences in duration and amount of alcohol consumed.

EARLIEST DETECTABLE CHANGE

In order to assess the earliest detectable change by echocardiography, the echocardiographic parameters among the three alcoholic groups and controls were individually compared.

The group with the shortest duration of drinking (group 1) did not show any significant difference as compared to controls. The difference between group 2 and controls was significant for end diastolic volume index, LV mass index, isovolumic relaxation time and deceleration time. Comparison between alcoholics with the longest drinking history (group 3) showed a significant increase in posterior wall thickness and peak late transmitral flow velocity (A)and a decrease in E/A ratio apart from the differences as group 2 demonstrated.

This implies that the earliest change that occurred were an increased end diastolic volume and left ventricular mass which were then followed by development of impaired LV relaxation.

Lazarevic et al[39] reported that LV volume changes occurred earlier which was followed by an increase in LV mass and later on impaired diastolic function.

STUDY LIMITATIONS

Only male patients were selected due to rarity of female alcoholics in this part of the country. Hence, the observations made in this study are limited to males only.

The details of alcohol consumption as reported by the patients might be inaccurate as alcoholics tend to underestimate the quantity of alcohol consumed.

This study was performed in a cross sectional method. Hence, it cannot be conclusively said whether or not a chronic alcoholic would develop symptomatic diastolic dysfunction and later on systolic dysfunction if he/she continued to drink. This can be confirmed only by prospective studies.

SUMMARY

This study was an observational study which assessed the left ventricular function in 50 chronic asymptomatic alcoholics attending Stanley Government Hospital. They were compared with age matched controls. The alcoholics were divided into 3 groups to assess the correlation between the observed parameters and duration of alcoholism. The following observations were made in alcoholics:

- An increase in end diastolic volume index **($p < 0.001$)**
- An increase in left ventricular mass index **($p < 0.001$)**
- Increased posterior wall thickness **($p < 0.001$)**
- Prolonged isovolumic relaxation time and deceleration time **($p < 0.001$)**
- Increased peak late transmitral flow velocity (A) **($p < 0.001$)**
- Reduced E/A ratio **($p < 0.001$)**

There was no significant differences in the end systolic volume index, ejection fraction, interventricular septal thickness and peak early transmitral flow velocity (E) **($p > 0.05$).**

The earliest change involved the left ventricular volume and mass which was then followed by changes in the Doppler parameters implying mild left ventricular diastolic dysfunction. The increase in end diastolic volume, LV mass, posterior wall thickness and diastolic filling variables were more remarkable in alcoholics with longer duration of alcohol consumption.

Conclusions

CONCLUSIONS

- Preclinical cardiac abnormalities occur in chronic aymptomatic alcoholics which can be detected by echocardiography.

- The alcoholics showed features of impaired left ventricular relaxation with preserved ejection fraction.

- The changes observed in alcoholics by 2-D and M-mode echocardiography were increased left ventricular chamber volume in diastole, increased left ventricular mass and posterior wall thickness.

- The abnormalities in Doppler parameters were prolonged isovolumic relaxation time and deceleration time; increased peak late transmitral flow velocity and reduced E/A ratio.

- The earliest detectable change was an increase in LV chamber volume, mass and posterior wall thickness. This was then followed by impaired LV relaxation.

- The increase in LV volume, mass, posterior wall thickness and alteration in diastolic parameters were significantly related to the duration of drinking.

- The above findings can be considered as a marker for development of alcoholic cardiomyopathy but this cannot be validated unless longitudinal studies are carried out.

- Further studies are required to demonstrate the course and progression of these abnormalities and their reversibility with early abstinence.

Annexures

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PROFORMA

ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN CHRONIC ASYMPTOMATIC ALCOHOLICS

DEPARTMENT OF MEDICINE, SMCH

NAME –
AGE –
GENDER –
ADDRESS/CONTACT NO –

IP / OP NO -
DOA/DOD -

HISTORY :

SYMPTOMS –

PAST HISTORY-

SHT		PT	
DM		COPD/BA	
IHD		THYROID	
RHD		MEDICATIONS	

DSM IV CRITERIA :

Alcohol abuse	
Alcohol dependence	

PERSONAL HISTORY-

- Details of alcohol consumption :

Duration	
Frequency	
Amount	

- Smoker - Y / N

O/E : PR – Height - RS -

BP - Weight - CVS -

BSA - P/A-

INVESTIGATIONS :

*** CBC** *** FBS , PPBS**

*** RFT** *** F. LIPID PROFILE**

*** LFT** *** TFT**

*** ECG** *** CXR**

* **ECHO** : **2-D & M-mode measurements**

$LVID_d$ (cm)	
EDV (ml) / EDVI (ml/m^2)	
ESV (ml) / ESVI (ml/m^2)	
FS	
EF (%)	
PWT_d (cm)	
$IVST_d$ (cm)	
LVMI (g/m^2)	

Doppler LV diastolic filling variables

IVRT (ms)	
DT (ms)	
E (m/s)	
A (m/s)	
E/A	

MASTER CHART

S. NO.	NAME	AGE/SEX	DURATION OF ALCOHOLISM	AMOUNT OF ETHANOL	SMOKER	PR	BP	HEIGHT	WEIGHT	BSA	FBS/PPBS	HB	UREA/CREATININE	AST/ALT
			(years)	(grams/day)		(/min)	(mm Hg)	(cm)	(kg)	(m^2)	(mg/dl)	(g/dl)	(mg/dl)	(U/L)
1	SURESHKUMAR	28/M	6	100	YES	82	124/80	173	94	2.13	70 / 110	12	28/ 0.9	62 / 46
2	KARTHIKEYAN	34/M	11	120	NO	86	126/78	170	80	1.94	83 / 120	13.1	26 / 0.8	58 / 70
3	SUBAN SUBRAMANIAN	57/M	23	150	YES	76	130/82	160.9	78	1.87	76 / 124	13	25 / 0.8	74 / 67
4	RAMESH	46/M	15	130	NO	74	130/80	173	69	1.82	80 / 114	12.2	27 / 0.7	66 / 62
5	BALASUBRAMANIAN.B	38/M	10	110	YES	83	130/84	169	64	1.73	77 / 98	11.9	20 / 0.5	41 / 38
6	SELVAMANI.A	29/M	9	130	YES	79	120/78	172	73	1.87	85 / 117	13	22 / 0.6	42 / 40
7	SAGAYA PUSHPARAJ	45/M	15.5	180	NO	81	122/82	164	62	1.68	78 / 115	12.4	24 / 0.8	92 / 54
8	STEPHEN.S	24/M	7	160	YES	90	120/80	169	75	1.88	70 / 120	12.7	20 / 0.6	76 / 40
9	SURESH.R	45/M	18	200	YES	93	132/84	166	66	1.74	77 / 122	14	21 / 0.8	80 / 68
10	MANOHARAN.D	32/M	8	180	NO	89	120/70	174	85	2.03	84 / 99	12.9	18 / 0.7	63 / 38
11	DHINAKARAN.M	29/M	8	160	YES	77	130/70	167	52	1.55	89 / 118	12.4	24 / 0.9	54 / 43
12	RAJA.S	38/M	12	180	NO	72	132/80	152	54	1.51	75 / 110	11.6	28 / 1	60 / 42
13	HIREN N.DESAI	57/M	20	240	NO	84	118/70	176	115	2.37	104 / 124	12.5	28 / 0.9	100 / 78
14	KRISHNAKUMAR	34/M	10	120	YES	86	114/80	165	65	1.73	100 / 118	12.8	24 / 1.1	35 / 28
15	RAJESH	47/M	22	240	YES	75	120/80	172	82	1.98	76 / 120	13.2	23 / 0.7	86 / 64
16	GOKUL	32/M	9	90	NO	87	116/70	167	73	1.84	74 / 113	14	24 / 0.7	28 / 36
17	VIJAYAKUMAR	42/M	11	120	NO	76	120/80	171	85	2.01	82 / 116	13.5	26 / 0.9	68 / 72
18	CHANDRAN	42/M	12	120	YES	90	130/88	157	80	1.87	85 / 125	12	26 / 0.6	60 / 81
19	PARAMASIVAM	58/M	21	200	NO	79	130/76	154	76	1.8	80 / 108	13.8	27 / 1.1	98 / 73
20	SANJEEVI KUMAR	40/M	12	120	NO	82	124/80	176	90	2.1	79 / 116	13	26 / 0.8	70 / 40
21	SUNDARRAJ	51/M	19	120	YES	85	114/70	181	86	2.08	100 / 124	12.4	25 / 0.5	65 / 38
22	MURUGAN	30/M	9	90	YES	82	120/80	163	45	1.43	74 / 98	12.8	21 / 0.7	43 / 35
23	BHARATHI	36/M	12	100	NO	75	110/70	161	45	1.42	70 / 118	12	20 / 0.7	84 / 74
24	KUMAR	37/M	11	150	YES	76	130/84	164	78	1.89	75 / 108	13	23/0.8	104 / 68
25	MURUGESAN	44/M	8.5	120	NO	84	130/80	160	70	1.76	94 / 134	12.4	27 / 0.6	58 / 43
26	KUMAR	44/M	6	90	YES	80	134/80	166	57	1.62	83 / 127	13.1	21 / 0.9	32 / 42
27	SIVAKUMAR	52/M	21	240	YES	81	120/70	170	89	2.05	83 / 132	12.9	16 / 0.8	122 / 85
28	PERUMAL	36/M	11	120	NO	93	120/84	170	72	1.84	78 / 120	12.5	18 / 0.7	82 / 75
29	MANIMARAN	29/M	8	120	YES	79	116/78	168	63	1.71	74 / 126	13.4	21 / 1	44 / 33
30	VIJAY	41/M	12	90	YES	86	110/70	162	60	1.64	78 / 110	11.9	22 / 0.9	66 / 71
31	SUJAN	43/M	9	100	NO	77	126/78	171	70	1.82	90 / 134	11.8	20 / 0.8	72 / 58
32	RAMESH	27/M	7	130	YES	68	130/82	153	60	1.6	88 / 128	12.3	17 / 0.6	60 / 40
33	ARUN	44/M	12	130	NO	90	116/70	160	63	1.67	89 / 137	12.6	19 / 0.8	52 / 38
34	SENGOTTUVEL	50/M	17	150	NO	83	120/70	165	70	1.79	83 / 125	12	15 / 0.6	94 / 74
35	ANAND	33/M	8	100	YES	75	126/78	172	71	1.84	74 / 120	11.7	23 / 0.8	26 / 35
36	JAIKUMAR	28/M	6	90	YES	77	134/80	169	69	1.8	74 / 118	13	24 / 1.1	54 / 60
37	ARUL	35/M	13	120	NO	85	128/78	158	65	1.69	72 / 105	12.7	21 / 0.6	55 / 72
38	KANNAPAN	42/M	16	160	NO	86	130/80	170	78	1.92	68 / 123	12.6	19 / 0.8	92 / 120
39	RAVI	42/M	8	100	YES	91	126/82	163	66	1.73	74 / 107	13	17 / 0.6	76 / 55
40	MANIGANDAN	37/M	12	120	YES	85	126/74	168	83	1.97	91 / 128	11.8	16 / 0.8	68 / 45
41	VIJAYKUMAR	51/M	19	200	YES	74	130/76	176	88	2.07	80 / 130	12.7	18 / 0.9	88 / 43
42	RAMAIYAH	50/M	17	210	NO	84	120/80	159	60	1.63	87 / 129	12.5	17 / 0.8	93 / 38
43	KASIRAJA	45/M	9	110	NO	73	120/78	171	75	1.89	94 / 130	12	22 / 1	45 / 32
44	RAMAJEYAM	40/M	10	120	NO	65	128/74	165	69	1.78	76 / 104	12.4	21 / 0.8	81 / 58
45	MUNISEKAR	38/M	11	120	YES	66	126/72	169	58	1.65	95 / 128	11.8	18 / 0.7	67 / 65
46	SENTHIL	34/M	8	100	NO	95	120/80	154	53	1.51	86 / 119	12.8	17 / 0.8	55 / 73
47	MURALI	27/M	7	90	NO	85	130/80	157	62	1.64	81 / 132	12	19 / 0.7	60 / 70
48	MUTHUKUMAR	37/M	11	130	NO	69	132/84	165	71	1.8	79 / 128	12.4	17 / 0.7	81 / 63
49	SIVABALAN	32/M	10	120	YES	72	124/74	172	74	1.88	78 / 130	12.7	19 / 0.6	73 / 54
50	MANI	47/M	19	180	YES	74	120/78	161	59	1.62	77 / 125	13.6	18 / 0.7	75 / 96

MASTER CHART

S. NO.	NAME	AGE/SEX	DURATION OF ALCOHOLISM	AMOUNT OF ETHANOL	SMOKER	PR	BP	HEIGHT	WEIGHT	BSA	FBS/PPBS	HB	UREA/CREATININE	AST/ALT
			(years)	(grams/day)		(/min)	(mm Hg)	(cm)	(kg)	(m^2)	(mg/dl)	(g/dl)	(mg/dl)	(U/L)
51	MATHIVANAN	47/M	—	—	NO	80	112/70	170	89	2.05	82 / 130	11.9	21 / 0.9	31 / 32
52	BALASUBRAMANIAM	53/M	—	—	YES	88	130/80	171	70	1.82	77 / 102	12.4	18.6/1.1	26 / 26
53	SHEKAR	54/M	—	—	YES	64	134/84	170	72	1.84	70 / 123	12.8	18 / 0.7	21 / 35
54	BASIL P.T	29/M	—	—	NO	86	114/66	153	60	1.6	75 / 127	12.4	17 / 0.8	24 / 32
55	BASKER	44/M	—	—	NO	71	122/86	168	63	1.71	76 / 124	12	19 / 1	32 / 36
56	KANAGARAJ	25/M	—	—	NO	80	120/80	160	63	1.67	84 / 130	13.2	17 / 0.9	27 / 21
57	THANGAVEL	50/M	—	—	YES	74	132/80	162	60	1.64	83 / 134	12.8	21 / 0.8	36 / 29
58	JAGADISH	29/M	—	—	NO	81	120/70	172	68	1.8	70 / 129	13.1	19 / 0.9	26 / 25
59	SURESH	26/M	—	—	NO	78	120/80	152	48	1.42	78 / 124	13.2	21 / 0.9	21 / 18
60	JEGANMOHAN	32/M	—	—	YES	83	118/70	160	55	1.56	76 / 124	12.8	22 / 1.1	32 / 37
61	RAMESH	43/M	—	—	YES	89	124/78	169	58	1.65	73 / 128	12.4	17 / 0.7	27 / 31
62	KATHIRAVEL	36/M	—	—	NO	90	134/82	172	63	1.73	80 / 127	12.6	19 / 0.8	25 / 18
63	SRIRAMULU	29/M	—	—	NO	80	132/80	163	57	1.61	84 / 124	12.8	21 / 0.9	28 / 32
64	VENKATESH	41/M	—	—	YES	76	114/78	157	52	1.51	80 / 120	11.8	22 / 0.9	40 / 29
65	SUBBU	44/M	—	—	YES	70	116/78	159	50	1.49	77 / 119	13.2	19 / 0.8	30 / 20
66	ALEX	55/M	—	—	NO	66	120/70	163	60	1.65	79 / 134	13.4	19 / 0.9	34 / 27
67	DINESH	37/M	—	—	NO	75	128/80	172	75	1.89	84 / 124	13	20 / 0.8	21 / 24
68	SUBBAIYAH	49/M	—	—	YES	78	132/84	173	80	1.96	82 / 124	12	21 / 0.8	16 / 19
69	VENUGOPAL	52/M	—	—	NO	87	130/74	169	70	1.81	72 / 116	12.6	22 / 08	27 / 18
70	SRIRAM	34/M	—	—	NO	88	124/86	166	65	1.73	66 / 118	12.5	24 / 1	21 / 25
71	SUNDAR	26/M	—	—	YES	82	126/74	164	59	1.64	65 / 104	12.8	21 / 0.8	29 / 34
72	MURUGAVEL	33/M	—	—	NO	94	126/78	170	75	1.88	70 / 112	13	23 / 0.9	23 / 31
73	RAJKUMAR	42/M	—	—	NO	80	122/84	169	65	1.75	74 / 128	13.1	23 / 1	28 / 24
74	VELU	49/M	—	—	YES	86	128/84	172	79	1.94	78 / 134	12.9	22 / 0.9	29 / 31
75	VENKATAIYAH	37/M	—	—	YES	78	120/82	168	73	1.85	80 / 126	12.8	18 / 0.9	35 / 38
76	DINESHKUMAR	28/M	—	—	NO	68	124/74	158	60	1.62	84 / 124	12.7	19 / 0.7	26 / 31
77	SUBRAMANI	54/M	—	—	NO	70	120/74	161	72	1.79	94 / 132	11.9	21 / 0.7	19 / 14
78	ARAVIND	40/M	—	—	YES	74	118/80	155	62	1.63	77 / 118	12.1	20 / 0.9	18 / 25
79	BALAJI	31/M	—	—	NO	76	110/78	165	73	1.83	78 / 125	12.4	19 / 0.8	23 / 27
80	MUKUNDAN	35/M	—	—	YES	86	120/70	167	76	1.88	90 / 130	12.3	18 / 0.8	31 / 25
81	KARTHIK	45/M	—	—	YES	82	120/78	158	70	1.75	92 / 128	12.6	17 / 0.9	29 / 34
82	DURAISAMY	50/M	—	—	YES	84	130/78	173	80	1.96	75 / 120	12	16 / 0.6	24 / 32
83	KANAKARAJ	48/M	—	—	YES	68	130/84	171	75	1.89	79 / 125	13	17 / 0.8	37 / 29
84	KANNAN	26/M	—	—	NO	74	126/82	163	68	1.75	88 / 138	13.1	24 / 0.9	35 / 26
85	RAMAN	32/M	—	—	NO	78	122/70	160	57	1.59	83 / 134	13.2	23 / 1.1	40 / 36
86	KUPPUSAMY	31/M	—	—	YES	80	118/78	153	58	1.57	82 / 120	13.6	21 / 0.9	30 / 32
87	BHOOPATHI	43/M	—	—	NO	85	124/80	169	72	1.84	78 / 115	14	19 / 0.9	23 / 30
88	MURUGAN	47/M	—	—	NO	82	126/76	164	80	1.91	76 / 109	12.9	18 / 0.7	25 / 29
89	SURESHKUMAR	38/M	—	—	YES	88	112/64	170	77	1.91	69 / 110	13	17 / 0.9	28 / 31
90	JOHN	34/M	—	—	YES	79	110/74	158	63	1.66	68 / 124	13.9	18 / 0.7	26 / 34
91	BHOOPALAN	29/M	—	—	NO	75	120/84	162	71	1.79	75 / 126	13	19 / 1	18 / 21
92	ARISTO	30/M	—	—	NO	68	124/72	159	68	1.73	77 / 123	12.7	19 / 0.8	18 / 11
93	SATHIYA	36/M	—	—	NO	67	110/76	171	78	1.92	86 / 120	12	17 / 0.7	16 / 19
94	BABU	27/M	—	—	YES	85	132/84	172	81	1.97	83 / 138	11.8	16 / 0.6	25 / 29
95	PAUL	51/M	—	—	YES	86	118/82	179	88	2.09	74 / 129	13	21 / 0.9	18 / 23
96	RAMKUMAR	48/M	—	—	NO	79	126/80	166	60	1.66	81 / 127	12.4	20 / 0.8	19 / 24
97	VENKATRAMAN	42/M	—	—	YES	70	130/68	164	63	1.69	80 / 131	12.7	19 / 0.8	18 / 15
98	KRISHNAN	36/M	—	—	NO	82	114/78	172	76	1.91	75 / 123	12.9	17 / 0.6	22 / 31
99	GOVINDASAMY	46/M	—	—	YES	83	120/84	169	73	1.85	70 / 126	12.8	19 / 0.8	23 / 19
100	RAMASAMY	40/M	—	—	YES	85	110/70	168	61	1.69	82 / 132	13.1	20 / 0.8	27 / 32

MASTER CHART

GGT	T.BILIRUBIN	T.CHOLESTEROL	TGL / LDL	TSH	ECG	CXR	LVID (d)	EDVI	ESVI	FS	EF	PWT (d)	IVST (d)	LVMI	IVRT	DT	E	A	E/A
(U/L)	(mg/dl)	(mg/dl)	(mg/dl)	(µIU/ml)			(cm)	(ml/m^2)	(ml/m^2)	(%)	(%)	(cm)	(cm)	(g/m^2)	(ms)	(ms)	(m/s)	(m/s)	
48	1.1	164	120 / 65	0.98	WNL	NAD	5	43.66	11.73	35	73	1	1.2	81.89	76	160	1	0.91	1.09
72	0.9	134	98 / 46	3.45	WNL	NAD	4.9	56.19	13.4	50	87	0.9	1	84.7	86	182	0.9	0.65	1.38
93	0.7	178	112 / 78	2.98	WNL	NAD	4.5	62.03	17.11	39	78	1.2	1	92.6	102	221	0.5	0.74	0.67
78	0.8	163	132 / 65	3.61	WNL	NAD	4.4	61.53	15.93	35	72	1	1.1	86.93	90	196	0.7	0.59	1.18
64	1	127	88 / 43	1.02	WNL	NAD	4.3	52.02	13.73	41	80	1	0.9	82.36	88	170	0.63	0.57	1.1
52	1.2	146	110 / 50	0.69	WNL	NAD	4.2	42.78	15.6	46	84	1.1	1	78.29	70	163	0.94	0.62	1.51
79	0.9	128	99 / 60	1.39	WNL	NAD	4.7	52.98	13.36	42	81	1	0.9	91.32	86	172	0.71	0.92	0.77
58	0.8	187	140 / 88	4.01	WNL	NAD	4.2	40.95	12.23	43	81	0.9	1	67.66	72	176	1.02	0.74	1.37
86	0.6	156	112 / 58	2.58	WNL	NAD	4.9	48.85	16.09	48	78	1	0.8	87.9	90	202	0.94	1.02	0.92
63	0.7	138	132 / 74	3.62	WNL	NAD	4.5	44.33	14.28	34	71	0.9	0.9	65.43	82	140	0.98	0.58	1.68
71	1	129	120 / 80	3.87	WNL	NAD	4	56.77	14.19	42	81	0.9	1	76.27	79	136	0.83	0.57	1.45
41	1	150	78 / 68	2.49	WNL	NAD	4.5	60.92	15.89	41	80	0.8	1	87.96	80	163	0.82	0.74	1.1
104	0.9	170	110 / 60	1.58	WNL	NAD	5.2	53.16	13.5	47	78	1.1	1.1	93.15	94	217	0.61	0.5	1.22
45	0.8	147	126 / 87	3.72	WNL	NAD	4.4	46.82	15.02	45	77	0.8	1.1	79.63	83	143.5	0.91	0.67	1.36
87	0.8	181	134 / 78	1.09	WNL	NAD	4.8	54.04	16.71	36	80	1	1.1	91.87	120	196	1.03	0.94	1.09
74	1	193	125 / 65	2.05	WNL	NAD	4.2	44.56	12.17	52	84	0.8	1.1	69.46	81	174	0.58	0.49	1.18
66	1.1	139	128 / 67	2.78	WNL	NAD	5	47.76	12.93	42	73	0.8	0.9	73.05	91	186	0.75	0.73	1.02
93	0.8	142	140 / 80	4.02	WNL	NAD	4.8	47.59	15.5	45.2	77	0.9	1	84.93	74	189	0.66	0.72	0.92
48	0.9	138	136 / 76	4.22	WNL	NAD	4.9	54.45	16.83	43.5	75	1.1	1	104.5	110	190	0.7	1.01	0.69
72	1	162	128 / 76	3.94	WNL	NAD	4.8	49.52	13.33	39	70	0.9	1	75.63	100	180	0.69	0.54	1.27
83	0.9	170	125 / 79	2.46	WNL	NAD	5.1	48.56	15.38	36	67	1	1.1	96.53	114.8	186.5	0.7	0.9	0.78
54	0.8	158	130 / 81	3.19	WNL	NAD	4.4	48.95	18.18	24	65	0.9	0.8	82.5	76	138	0.53	0.44	1.2
58	0.9	148	115 / 72	2.56	WNL	NAD	4.4	57.04	14.08	51	85	1	0.8	83.03	85	191	1.03	0.89	1.15
74	0.8	125	108 / 60	0.98	WNL	NAD	4.5	46.03	12.69	43.8	75	1	1.1	86.76	87	150	0.9	0.7	1.28
81	0.5	163	129 / 64	1.68	WNL	NAD	4.3	56.7	19.26	36	66	1	1	80.96	93	161	0.83	0.6	1.38
56	0.7	178	101 / 65	3.52	WNL	NAD	4.5	40.12	14.19	41	72	0.7	0.8	64.5	77	153	0.6	0.4	1.5
81	0.9	145	119 / 59	2.58	WNL	NAD	5	58.05	17.56	40	72	1	1.2	101.05	100	226	0.76	0.89	0.85
79	1.2	184	123 / 67	4.03	WNL	NAD	4.4	50	14.67	36	81	0.8	1.1	74.87	86	172	0.8	0.74	1.08
67	1	174	129 / 83	3.86	WNL	NAD	4.5	48.54	14.03	53	77	0.8	0.9	71.97	76	153	0.9	0.79	1.13
72	0.6	163	130 / 78	2.57	WNL	NAD	4.6	53.04	15.24	33	69	0.9	0.9	73.67	94	180	0.83	0.68	1.22
83	0.8	156	127 / 70	2.94	WNL	NAD	4.7	50.8	13.95	42	80	0.8	1.1	84.29	72	164	1.02	0.77	1.32
67	0.9	176	124 / 73	3.15	WNL	NAD	4.2	47.68	13.13	47	74	0.7	0.9	63.3	74	147	0.75	0.47	1.59
86	0.7	144	112 / 64	0.99	WNL	NAD	4.5	49.9	14.37	50	73	1	1	91.78	99	171	0.69	0.6	1.15
96	1	185	133 / 82	1.72	WNL	NAD	4.7	52	15.64	37	85	1.1	0.8	85.7	75	192	0.81	0.72	1.13
62	1.1	182	135 / 79	2.95	WNL	NAD	4.7	47.83	14.13	48	78	0.8	0.9	71.91	80	132	0.75	0.57	1.31
46	0.4	136	126 / 63	2.04	WNL	NAD	5	43.89	13.33	41	73	0.7	0.9	75.44	73	139.5	0.83	0.58	1.43
61	0.9	129	132 / 70	1.65	WNL	NAD	4.5	50.89	15.98	39	67	1	1	90.69	88	142	0.94	0.92	1.02
72	0.8	147	121 / 67	1.9	WNL	NAD	4.9	51.04	14.58	40	69	1.1	1	97.96	82	188	0.54	0.68	0.79
53	1	183	142 / 89	3.74	WNL	NAD	4.4	48.43	13.29	36	72	0.9	1.1	85.45	77	157	0.98	0.86	1.13
68	0.7	172	130 / 82	2.16	WNL	NAD	5.1	47.72	14.72	44	75	0.9	1.1	95.43	93	179	0.75	0.77	0.97
79	0.8	189	129 / 73	0.86	WNL	NAD	5.2	57.97	15.46	45	68	1.1	0.7	81.63	102	208	0.78	0.94	0.82
84	0.6	190	128 / 74	0.97	WNL	NAD	4.9	54.6	15.33	38	73	0.9	1	100.2	94	185	0.93	0.89	1.04
75	0.7	157	125 / 65	1.58	WNL	NAD	4.8	52.38	13.76	47	78	0.9	1	84.03	73	185	0.67	0.46	1.45
55	0.9	164	130 / 76	4.01	WNL	NAD	4.5	48.54	15.17	41	81	1	0.9	80.27	81	176.5	0.83	0.86	0.96
64	1	178	127 / 76	2.47	WNL	NAD	4.2	52.72	15.51	35	77	0.9	0.8	66.57	83	165	0.6	0.52	1.15
71	0.8	184	128 / 82	2.38	WNL	NAD	4	50.33	13.24	29	78	0.8	1	72.64	74	147	0.74	0.54	1.37
63	0.6	166	123 / 74	3.42	WNL	NAD	4.3	45.12	12.19	30	70	0.8	1	75.18	76	136	0.95	0.61	1.55
58	0.8	173	118 / 69	2.89	WNL	NAD	4.5	51.94	14.45	42	68	0.9	1.1	85.15	85	158	0.77	0.59	1.3
67	0.7	168	113 / 61	1.73	WNL	NAD	4.4	48.4	13.82	38	71	1	0.9	73.27	82	167	1.03	0.83	1.24
102	0.8	152	124 / 65	2.94	WNL	NAD	4.2	65.43	18.51	43	75	1.2	1.1	103.36	96	198	0.86	1.12	0.76

MASTER CHART

GGT	T.BILIRUBIN	T.CHOLESTEROL	TGL / LDL	TSH	ECG	CXR	LVID (d)	EDVI	ESVI	FS	EF	PWT (d)	IVST (d)	LVMI	IVRT	DT	E	A	E/A
(U/L)	(mg/dl)	(mg/dl)	(mg/dl)	(µIU/ml)			(cm)	(ml/m^2)	(ml/m^2)	(%)	(%)	(cm)	(cm)	(g/m^2)	(ms)	(ms)	(m/s)	(m/s)	
17	0.6	185	131 / 79	3.21	WNL	NAD	4.6	47.32	12.68	41	72	1	1.2	88.4	127	220	0.86	0.7	1.23
36	0.6	149	132 / 77	1.07	WNL	NAD	4.6	54.39	17.03	38	68	0.9	1	81.38	96	130	0.9	0.8	1.125
29	0.4	179	126 / 73	2.06	WNL	NAD	4.7	57.07	14.67	43	74	1	1.1	101.92	84	200	0.8	0.9	0.89
38	1.1	157	123 / 75	3.68	WNL	NAD	4.2	50.63	14.37	39	70	0.9	0.9	74.17	63	140	0.9	0.6	1.5
19	0.9	169	137 / 82	1.82	WNL	NAD	4	42.69	12.68	38	68	0.9	1.1	69.4	90	127	1	0.8	1.25
17	1	150	132 / 74	2.97	WNL	NAD	4	38.92	13.77	35	65	0.9	0.9	65.68	101	136	0.7	0.5	1.4
25	0.6	138	131 / 72	2.78	WNL	NAD	4.4	54.88	15.24	41	72	0.9	1	84	76	160	1.1	0.8	1.375
27	0.8	142	127 / 68	3.25	WNL	NAD	4.4	48.33	11.11	41	76	0.8	1	79.12	120	113	0.76	0.4	1.9
21	1.1	146	124 / 66	2.14	WNL	NAD	4.3	52.82	16.9	40	79	0.9	0.9	86.83	74	150	0.96	0.8	1.2
33	0.9	157	134 / 68	2.76	WNL	NAD	4.6	42.95	12.82	37	81	0.8	0.8	75.58	80	167	0.77	0.61	1.26
23	0.7	173	125 / 67	0.96	WNL	NAD	4.5	46.06	13.33	42	74	0.9	1	86.59	74	145	0.86	0.53	1.62
27	0.9	179	119 / 70	1.49	WNL	NAD	4.3	39.88	13.87	39	85	0.8	1.1	76.73	86	157	0.89	0.7	1.27
45	1	160	117 / 64	2.48	WNL	NAD	4.1	44.72	15.53	37	65	1	0.9	76.38	73	163	1	0.58	1.72
34	0.5	154	132 / 71	3.21	WNL	NAD	3.8	41.72	15.23	52	72	0.8	1	66.93	92	144	0.63	0.75	0.84
31	0.6	183	129 / 72	4.09	WNL	NAD	4	47.65	12.75	44	82	0.9	0.8	68.08	101	170	0.86	0.74	1.16
23	0.9	157	121 / 61	3.65	WNL	NAD	4.1	51.51	14.55	39	71	0.8	0.9	63.99	98	220	0.92	0.69	1.33
25	1	164	127 / 69	2.97	WNL	NAD	4.5	44.44	14.29	40	70	0.7	1	65.12	81	140	0.65	0.72	0.9
35	0.8	171	128 / 63	2.43	WNL	NAD	5.2	52.55	16.32	48	78	0.8	1.1	99.05	80	166	1.02	0.53	1.92
29	0.7	183	133 / 72	2.79	WNL	NAD	4.7	50.28	15.46	39	67	0.9	0.8	73.11	70	183	0.97	0.64	1.51
37	0.6	186	139 / 82	3.98	WNL	NAD	4.7	47.51	14.45	42	81	0.9	1	88.68	67	197	0.88	0.53	1.66
24	0.4	159	129 / 72	3.64	WNL	NAD	4.2	47.56	15.85	41	70	0.9	0.8	66.97	88	130	0.78	0.65	1.2
18	1	161	134 / 65	3.01	WNL	NAD	4.2	47.34	12.77	37	86	0.8	1	63.12	74	151	0.86	0.53	1.62
31	1.1	157	109 / 59	2.05	WNL	NAD	4.3	53.71	15.43	38	72	1	0.9	75.85	75	146	0.92	0.5	1.84
25	0.7	153	118 / 64	2.38	WNL	NAD	4.4	45.36	12.89	42	80	0.7	1.1	65.98	69	187	0.77	0.63	1.22
27	0.8	174	123 / 62	1.73	WNL	NAD	4.1	55.68	16.76	29	78	0.9	0.9	61.69	72	134	0.65	0.72	0.9
35	1.2	187	114 / 67	1.9	WNL	NAD	4	55.56	14.2	40	82	1.1	1	84.07	81	147	1.02	0.64	1.59
21	0.9	175	126 / 65	2.43	WNL	NAD	4.6	48.6	11.73	35	76	0.8	0.9	71.31	85	153	0.93	0.87	1.06
17	0.6	168	142 / 83	2.09	WNL	NAD	5.2	44.79	13.5	37	82	0.8	0.8	89.09	93	164	0.86	0.62	1.38
19	0.7	155	137 / 71	3.49	WNL	NAD	5	39.35	13.11	41	80	1	0.8	86.45	100	170	0.9	0.71	1.27
14	1.1	163	129 / 69	3.12	WNL	NAD	4.7	44.68	14.89	39	67	1	0.7	70.38	84	144	1	0.85	1.17
23	0.9	176	139 / 84	2.38	WNL	NAD	4.7	40.57	18.86	40	72	0.8	0.9	75.61	75	140	0.7	0.52	1.35
16	0.8	182	129 / 58	2.34	WNL	NAD	4.8	38.27	15.31	45	74	0.8	1.1	81.03	79	168	0.64	0.81	0.79
27	1	158	112 / 72	0.84	WNL	NAD	4.4	42.86	13.76	50	77	0.9	1	72.89	80	180	0.69	0.53	1.3
36	0.6	175	134 / 78	1.27	WNL	NAD	3.8	39.42	14.28	38	81	1.2	0.9	71.9	90	201	0.88	0.6	1.47
41	0.7	163	109 / 62	1.83	WNL	NAD	4.2	60.38	13.83	42	70	0.8	1.1	80.38	92	183	0.97	0.62	1.56
37	0.8	173	108 / 62	2.04	WNL	NAD	3.9	51.59	12.1	35	69	0.9	1	72.34	112	174	0.9	0.49	1.84
29	1.2	148	126 / 82	2.05	WNL	NAD	4.1	41.3	12.5	41	71	0.8	0.9	57.38	74	162	0.87	0.55	1.58
32	1	187	132 / 66	3.72	WNL	NAD	4	40.31	16.23	43	73	0.9	0.8	53.1	73	157	0.76	0.66	1.15
25	0.8	191	142 / 83	3.07	WNL	NAD	4.8	34.55	13.61	38	65	1	0.8	77.37	83	154	0.84	0.89	0.94
22	0.6	153	108 / 53	1.96	WNL	NAD	4.2	49.4	12.65	40	79	0.9	1	76.99	76	140	0.77	0.58	1.32
18	0.8	154	115 / 64	1.34	WNL	NAD	4.5	43.58	11.17	37	80	0.8	0.8	63.48	79	166	0.75	0.82	0.91
15	0.7	167	127 / 76	1.28	WNL	NAD	4.4	39.3	14.45	28	72	1	0.9	79.63	82	182	1.12	0.84	1.33
27	0.9	178	141 / 92	2.36	WNL	NAD	4.7	52.08	14.58	45	75	1.1	0.7	74.33	86	196	0.81	0.77	1.05
31	0.6	162	135 / 81	2.15	WNL	NAD	4.5	48.73	16.24	39	82	0.8	1	67.42	94	140	0.98	0.74	1.32
17	0.5	179	142 / 84	3.49	WNL	NAD	4.4	54.07	15.31	41	77	0.9	1.1	70.73	96	155	0.71	0.65	1.09
28	1.1	157	128 / 78	3.02	WNL	NAD	4.1	51.2	16.27	40	68	0.9	0.8	63.61	81	120	0.93	0.59	1.57
38	1	169	140 / 70	2.84	WNL	NAD	4.8	43.2	13.61	37	72	0.8	0.9	81.11	74	138	0.76	0.54	1.4
33	0.4	158	130 / 72	2.75	WNL	NAD	5	46.07	13.09	35	70	1	0.9	88.96	75	157	0.8	0.65	1.23
26	0.7	147	108 / 68	2.1	WNL	NAD	4.5	49.19	11.89	42	64	1.1	0.7	71.79	107	181	0.83	0.88	0.94
43	0.8	151	112 / 70	3.02	WNL	NAD	4.6	46.75	11.24	36	68	0.9	1	100.5	90	170	0.75	0.83	0.9

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Assessment of left ventricular function in chronic asymptomatic alcoholics

Principal Investigator : Dr.Priya Kubendran PG in MD(GM)

Designation : PG in MD(GM)

Department : Department of Medicine
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 18.04.2011 at the Modernized Seminar Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate form the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

Figure 1 : M – mode echocardiography at the level of the mitral valve

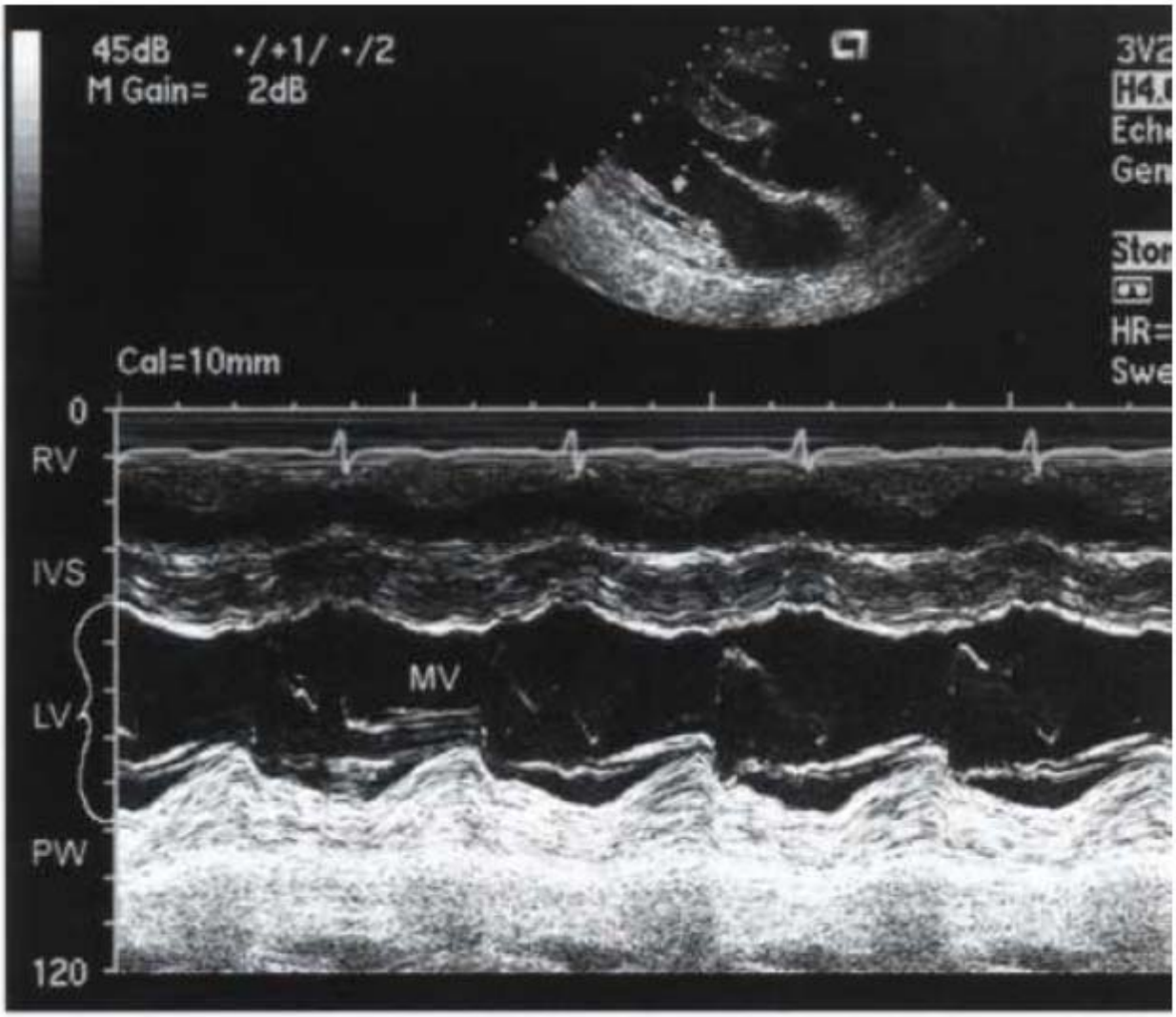


Figure 2 : 2 - D image of the heart, parasternal long axis view

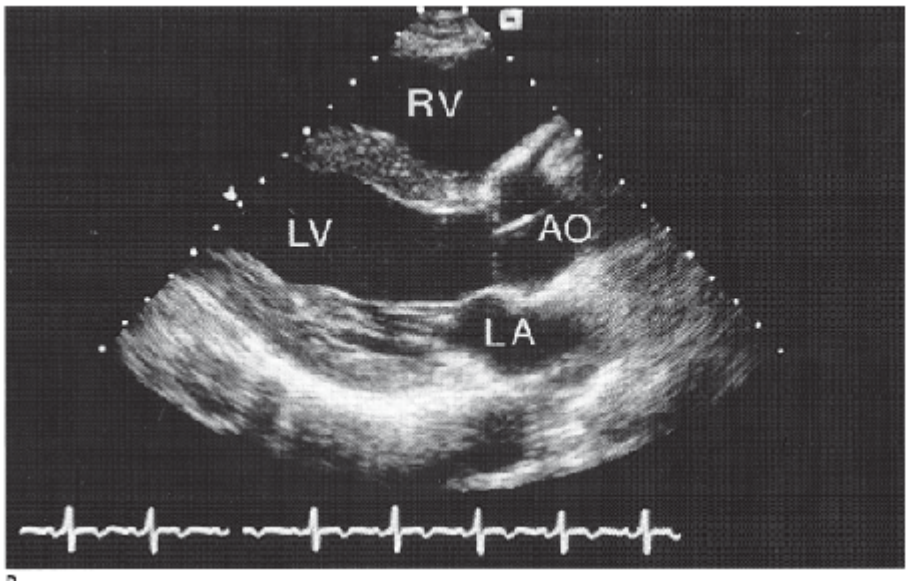


Figure 3 : Diastolic filling patterns

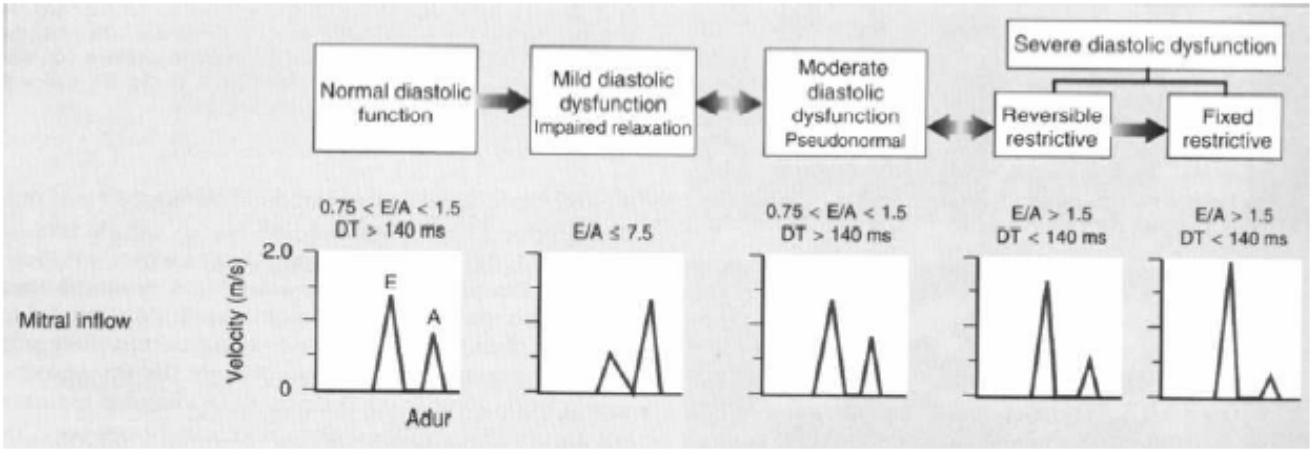


Figure 4 : Percentage of alcoholics in each group

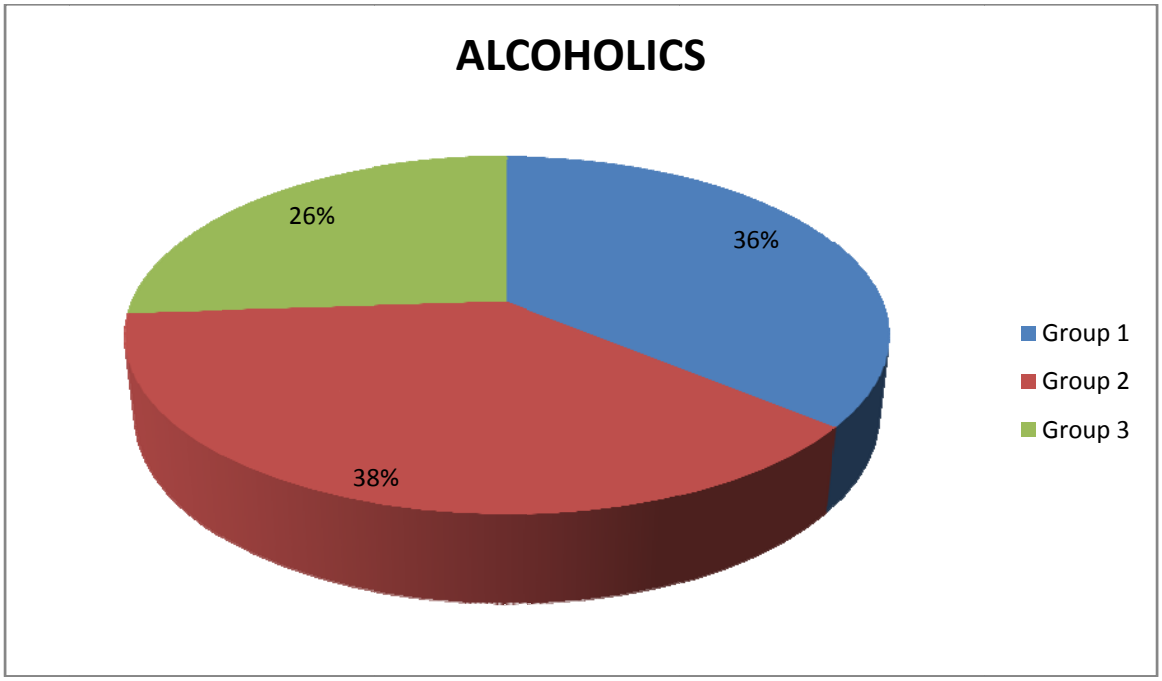
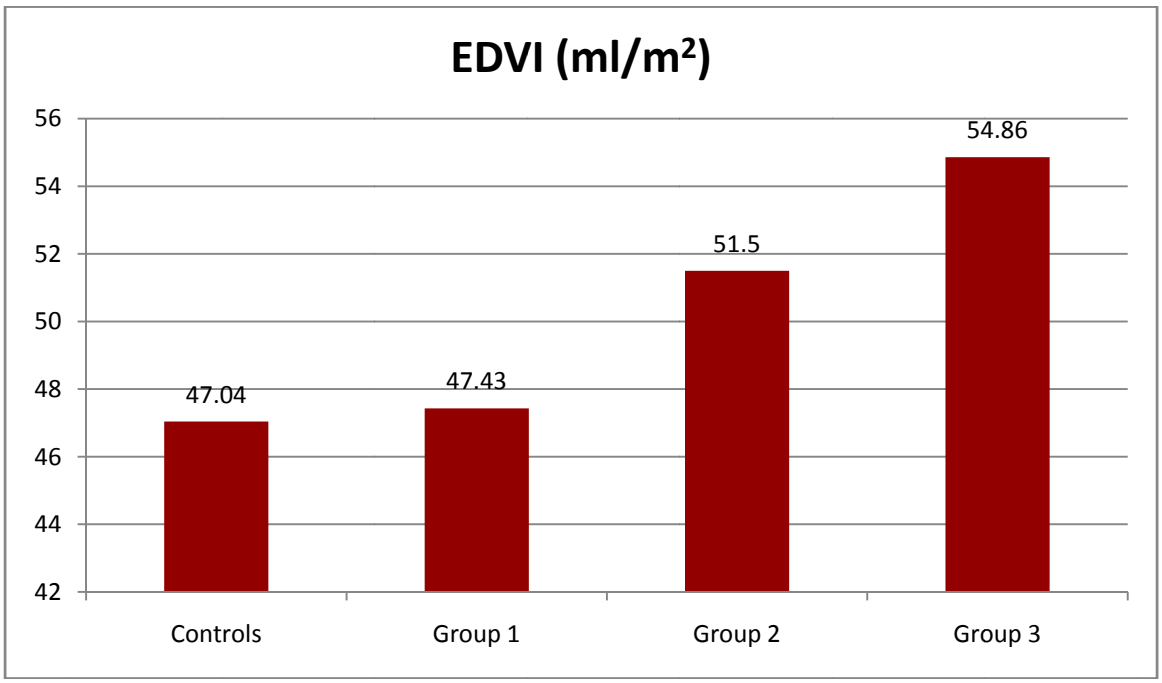


Figure 5 : Comparison of means of end diastolic & systolic volume indices between controls and alcoholics



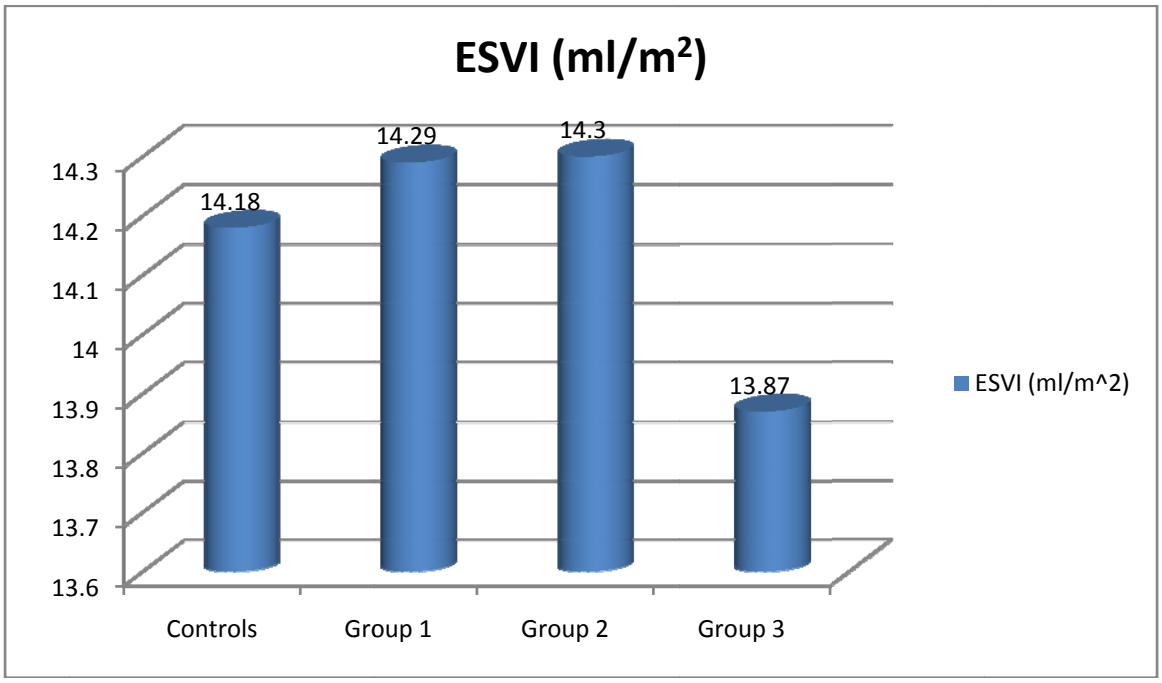
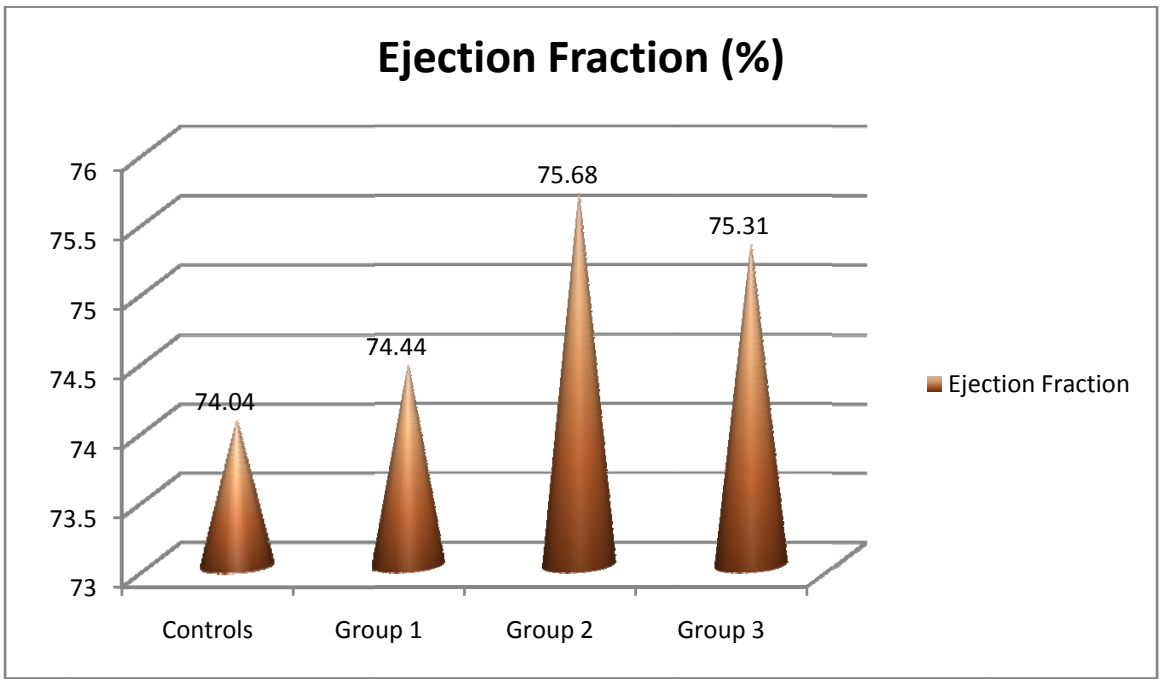


Figure 6 : Comparison of means of ejection fraction & LV mass index between controls & alcoholics



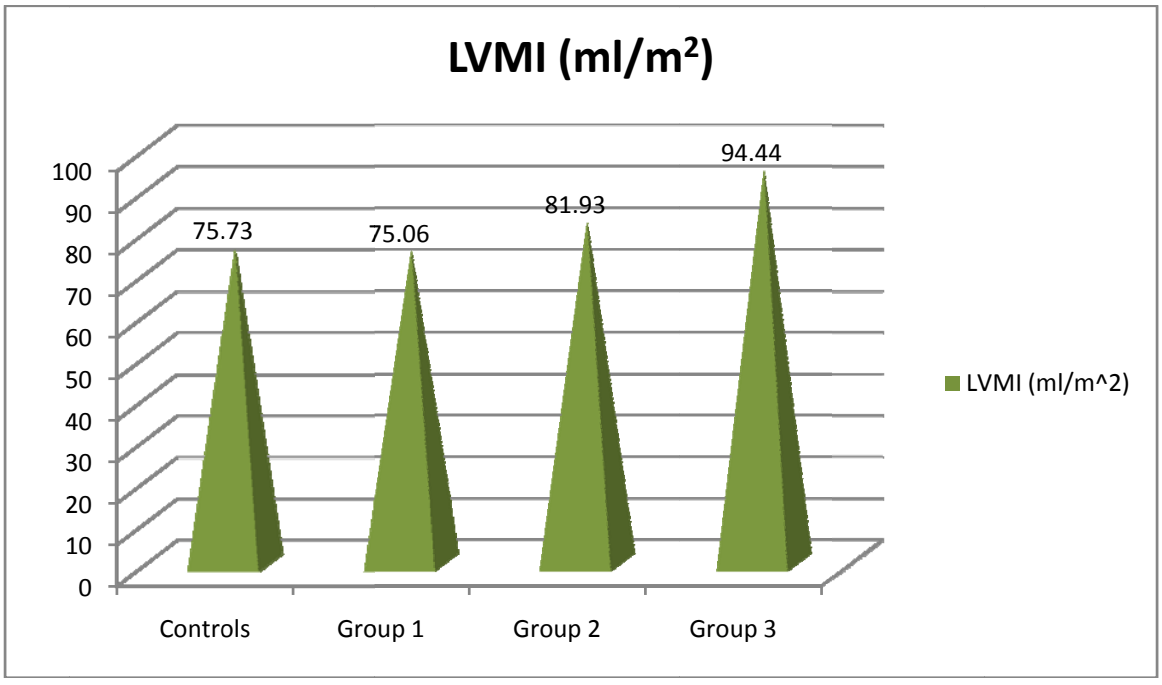


Figure 7 : Comparison of the means of posterior wall & interventricular septal thickness between controls and alcoholics

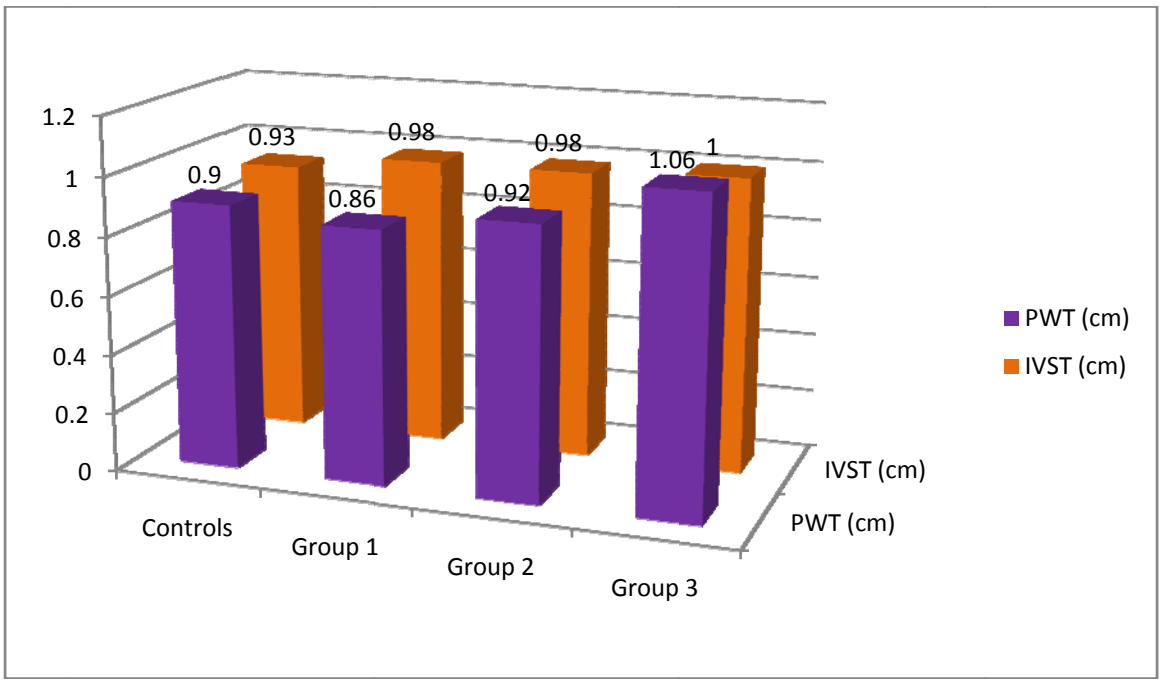
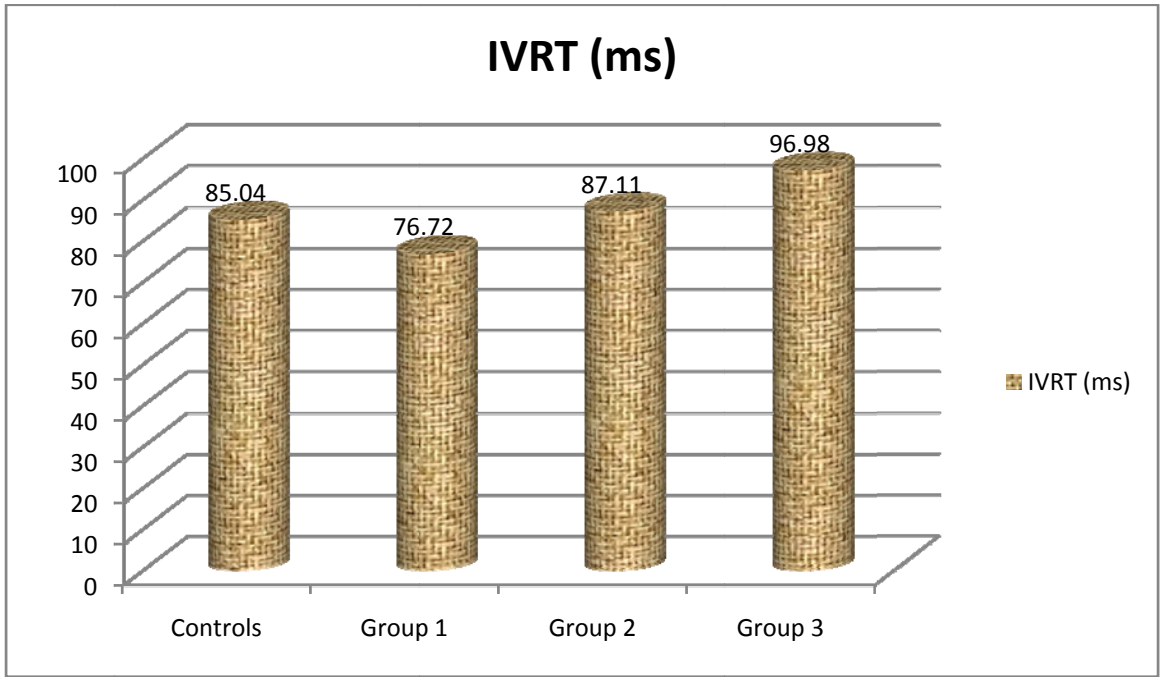


Figure 8 : Comparison of the means of isovolumic relaxation time & deceleration time between controls and alcoholics



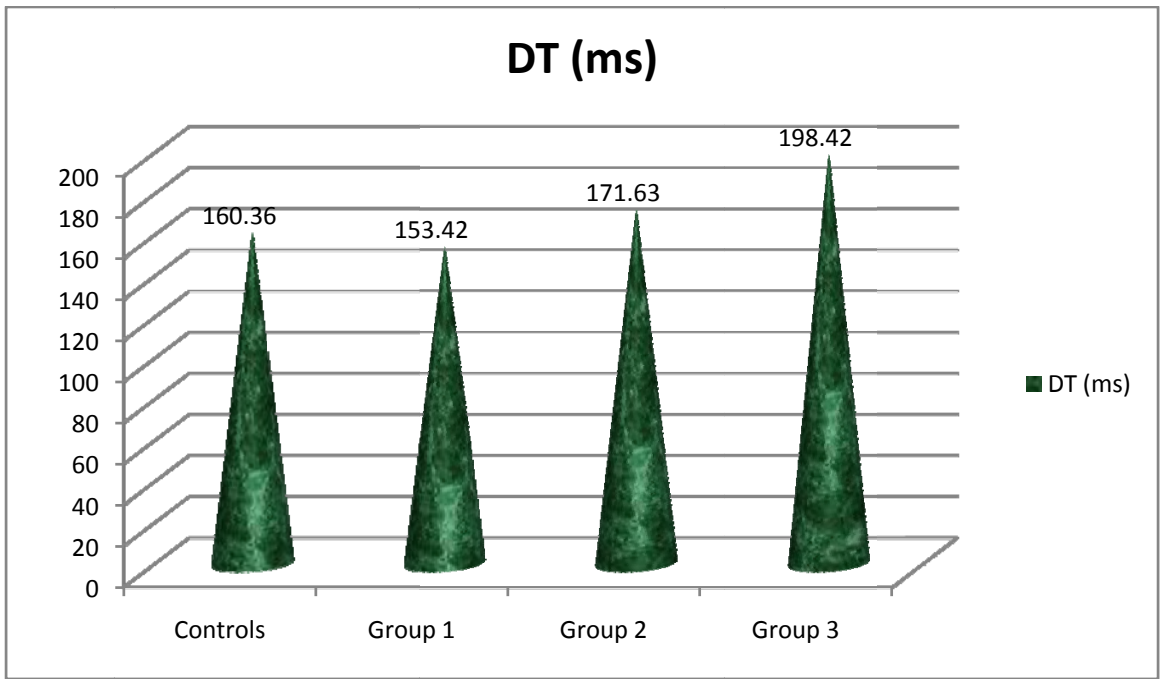


Figure 9 : Comparison of the means of the transmitral flow velocities between controls & alcoholics